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**THE DISEASES OF THE HEART AND OF
THE AORTA.**

THE
DISEASES OF THE HEART
AND OF
THE AORTA.

BY
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OF MILAN; ETC., ETC.

EIGHTY-ONE ILLUSTRATIONS.

PART II.

"Vaga enim experientia, et se tantum sequens, mera palpatio est. . . . At cum experientia lege certa procedet, seriatim et continenter, de scientiis aliquid melius sperari poterit."—*Novum Organum*.

PHILADELPHIA:
LINDSAY & BLAKISTON.
1875.

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a purely nervous phenomenon, and forbids the assumption of organic disease. Syncope, though primarily due to temporary failure of the heart, results immediately from anæmia of the brain. It usually occurs whilst some physical effort is being made which overtaxes the strength of the heart; or shortly afterwards, from the resulting fatigue. A very common cause is lifting the arms above the head, as in adjusting window curtains, hanging pictures, etc. I have repeatedly known syncope to occur where fatty degeneration of the heart existed, from the simple effort of walking in the streets. Of this, several examples will be found in the appended cases. Vertigo is an allied symptom, and due to the operation, but in a less degree, of the same cause; namely, failure of arterial circulation in the brain.

The remarkable symptoms of pseudo-apoplexy and syncopal convulsions, of which several examples are given in the list of cases, are the highest expression of cerebral anæmia. They are characterized by brief duration, frequent repetition, and recovery of the patient without paralysis. In one of Mr. Adams' cases, no less than twenty such fits occurred within the last seven years of the patient's life; and in another, four such occurred in one year. The features in these fits, which not unfrequently occur after a full meal, are usually pallid and tranquil. Sometimes, however, they are congested and livid, the difference being manifestly due to the existence in the latter contingency of spasm of the muscles of respiration generally, or of those of the constrictors of the glottis in particular. Mr. Adams is of opinion that superficial congestion may arise from obstruction at the heart, consequent upon suspension of its movements; and that to cerebral congestion arising from the same cause, may be due in many cases the apoplectic symptoms. I think, however, the two forms are primarily identical. The difference between the cases of simple syncope with pallor and loss of consciousness, and those of an apoplectiform character, with lividity of the face, turgescence of the veins of the neck, and stertorous breathing, are due to the fact, that in the latter, which are of a more aggravated character, the respiratory centre is completely anæmiated, and convulsions, with spasm of the glottis and suspended respiration, are the necessary consequences.

Rhythmical irregularity of breathing, under the form of alternate acceleration and suspension, with intermediate stages of ascending and descending transition from a state of the most aggravated dyspnoea to one of apnoea, is a very common symptom in connexion with fatty degeneration of the heart. This remarkable phenomenon was first described by Dr. Cheyne, in his report of the well known case already referred to (p. 601). He says: "For several days his breathing was irregular; it would entirely cease for a-quarter of a minute, then it would become perceptible, though very slow; then, by degrees, it became heaving and quick; and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration."*

Doctor Stokes was the next writer who specially adverted to this symptom, which he very appropriately designates "respiration of ascending and descending rhythm." He refers to it as "a form of respiratory distress peculiar to this affection (fatty degeneration of the heart), consisting of a period of apparently perfect apnoea, succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the commencement of another apnoeal period."

In describing the case of a gentleman, aged sixty years, who exhibited a systolic basic murmur, very loud at the top of the sternum, and who was suddenly attacked with this peculiar form of dyspnoea, which at first returned at irregular intervals, but was constant for the last two months of his life, he says: "After a period of suspended breathing, leading his attendants to believe that he was dead, a very feeble, indeed barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high, heaving, and even violent breathing was established, which would then subside until the next period of suspension. This was frequently a-quarter of a minute in duration." He adds: "I have little doubt that this was a case of weakened and probably fatty heart, with disease of the aorta."†

* *Dublin Hospital Reports*, vol. ii., p. 216, 1818.

† *Dublin Quarterly Journal*, vol. ii., new series, August, 1846.

In this opinion, although no opportunity was afforded for confirming it by dissection, he was most probably correct. I believe however, that this remarkable symptom, of which several typical examples will be found in the annexed cases, although in the great majority of examples associated with the twofold lesion mentioned by Dr. Stokes, depends, not upon the fatty state of the heart, but upon atheromatous change, dilatation, and loss of elasticity in the aorta. In numerous examples of veritable fatty transformation of the heart, verified by *post mortem* examination, included in my records, this symptom was *not* exhibited; but in these the above mentioned changes in the aorta had not taken place; whereas, of the many cases in which rhythmical irregularity of breathing was present, *not one* failed to present this condition of the aorta, combined with fatty degeneration of the heart, where the actual state of the organs was determined by dissection.

In illustration of the rhythm of this form of dyspnœa, I may select the three following examples.

A man, aged sixty-two years, was suddenly seized with dyspnœa whilst proceeding to his work. Since then he had been subject to these attacks, which were accompanied with the most aggravated suffering. He felt as if his breathing were about to cease. Whilst he lay in bed, his breathing would gradually become quick and gasping, till it attained a rate of 63 in the minute, and then as gradually subside till it ceased entirely, continuing suspended for twenty-two seconds. During the period of apnœa there *was no change* in the heart's action or radial pulse, in regard to rate or otherwise (see Case 51, James K.)

A man aged sixty years, was subject to dizziness and paroxysmal dyspnœa, during which there were alternately periods of apnœa and suspirious respiration, the former occupying twenty-four, and the latter twenty-six seconds, the transition being rather abrupt and occupying only about one second. The action of the heart and the pulse *underwent no change* during this revolution of breathing (see Case 52, James D.).

A man of large stature, a plumber by trade, of temperate habits, suffering from general dropsy and the symptoms and

signs of hypertrophy and fatty degeneration of the heart, dilatation and roughening of the aorta, and disease of both the aortic and mitral valve, exhibited in the most typical form respiration of this character. The following description of this phenomenon is taken from my notes, made when the case was under observation, February, 1872.

"There was a period of complete suspension of breathing, which, in the recumbent posture, lasted fifteen seconds; during its continuance the poor patient, worn out by want of sleep, seemed to enjoy complete respite from suffering, and passed into a calm slumber. Then a shallow inspiratory movement of the chest was observed; another, somewhat deeper, followed at an interval of about two seconds; respiration became now progressively deeper and more rapid, till after about a dozen acts it had attained the character of a paroxysm of great severity, accompanied by a deep and distressing moan. After about a dozen such quick respiratory efforts, extending over a period of as many seconds, moaning ceased, and the breathing became gradually less and less deep and rapid; till, at the end of about fifteen seconds, it was entirely suspended, and another and similar cycle was commenced. During the paroxysm of dyspnoea the heart's action was remarkably irregular." (See Case of C. D., aortic disease.)

Respiration of this character existed in 29 cases included in my Tables; viz., in 16 of Table V.; in 1 of Table VI.; and in 12 of Table VII.*

Amongst Dr. Quain's cases I have not found a single example of the kind recorded, although dyspnoea is represented as present in thirty-four instances.

Is fatty transformation of the muscular fibres of the heart necessary to the existence of this symptom? Dr. Stokes holds that it is, whilst I regard it as only accessory; the essential condition being, in my judgment, dilatation and loss of elasticity in the aorta, with which, however, fatty change of the heart is almost invariably associated.

Doctor Seaton Reid, of Belfast, published, in 1850, a case in which this symptom existed, and the muscular structure of the heart was found healthy. There was, however, dilated hyper-

* *Vide* p. 648, *et seq.*

trophy of the left ventricle, dilatation with atheromatous change of the aorta, and incompetence of the aortic and mitral valve.*

Doctor Head communicated to the Medical Society of the College of Physicians, in the session 1867-8, another instance of a heart free from fatty degeneration, where this symptom in a typical form had been exhibited, the lesions present being aortic patency and hypertrophy of the left ventricle.

Doctor James Little, in 1868,† published three cases, in each of which "ascending and descending" respiration existed. In one of these, that of a man of sixty-nine years, fatty degeneration of the heart was found, but no valvular disease. In another, who was aged seventy-two, the left ventricle was hypertrophied, the aortic valves slightly rigid but not incompetent, and the aorta atheromatous and calcareous; and in the third, who was also seventy-two years old, the left ventricle was much dilated and hypertrophied, the aortic valves thick and rigid, but competent, and the entire arch of the aorta was dilated and atheromatous. The muscular structure of the heart was absolutely free from fatty degeneration, as reported by Professor Macalister, a most competent authority.

Thus it would appear that fatty degeneration of the substance of the heart, though usually present, does not constitute a necessary condition of the existence of rhythmical irregularity of breathing; whilst, on the other hand, in all the examples of it which have come under my notice, or of which I have seen the report, the primary portion of the aorta has been atheromatous and dilated.

Doctor Seaton Reid was the first to observe and record a change in the rate of the heart and pulse, inversely as those of respiration. He stated that during the period of accelerated breathing, the pulse became invariably slow, increasing in rate during that of apnoea.

Doctor Little also observed this phenomenon in two of his cases. He says: "In a note made on the 14th August, it was recorded that during ten seconds of the breathing period, there were only six systoles of the heart, and these were generally

* *Dublin Hospital Gazette.*

† *Dublin Quarterly Journal of Medical Science*, No. xci.

irregular in rhythm and force, while, during ten seconds of the apnoeal period, there were fifteen; the sounds were indistinct." Again, in reference to his third case, he says: "From a note made two days after his admission, I find that the apnoeal period lasted ten seconds, and during that time nineteen radial pulses were counted. The respiratory acts occupied twenty-five seconds, and during ten seconds, counted off at the height of the paroxysm, thirteen systoles were felt at the wrist."

I have not, in any of my cases, met with this change in the rate of the heart and pulse; on the contrary, I have specially and repeatedly noted the unaltered rate and character of both, during the changes of rhythm and force constituting this remarkable phenomenon.

Doctor Little propounds* the following theory in explanation of this symptom: "In health, the right and left ventricles, though differing so much in the thickness of their walls, are equally competent for their duties; the right is able to fill the pulmonary capillaries as thoroughly as the left, with the aid of the other forces which contribute to the circulation, fills the systemic. But if an abnormal burden is imposed on the left, if rigid valves narrow its outlet, or permit the blood it discharges at each systole to fall back into its cavity, or if the arterial coats, their elasticity destroyed by disease, no longer help the heart; if the aorta, instead of taking charge of each wave of blood as it leaves the ventricle and propelling it onward by the steady recoil of its walls, is permanently dilated, and allows each portion of blood to remain in its ascending trunk, and so to impede the entrance of that which follows—under any of these conditions, the left heart, however hypertrophied, may be quite unable to rid itself of the blood as rapidly as it is supplied to it by the right ventricle. Blood would, therefore, accumulate in the left auricle, in the pulmonary veins, and in the capillaries of the lungs. That blood, having already absorbed as much oxygen as it required, would fail to produce that impression on the ultimate filaments of the pneumogastric which black blood does, and which impression is converted by the nervous centres into the motor impulse which produces breathing. Breathing would,

* *Loco citat.*

therefore, cease; and, inasmuch as the respiratory act seems to assist in carrying the blood to the left side of the heart, it would no longer be so over-stimulated by fresh supplies, and its contractions would become less frequent and more regular. After a few systoles, however, it would succeed in discharging the red blood collected in its cavities to such an extent that they could receive some of that which lay in the pulmonary veins and lungs. Space being thus gained, the black blood which the pulmonary artery contained would reach the capillaries of the lungs in amount proportionate to that of the arterial which had gone forward, and sufficient air would be drawn into the chest to aërate so much blood. That very act would carry forward a still larger charge of arterial blood to the left side, and make room for the reception, by the lungs, of a still further increase of venous blood; and, as a consequence, a still deeper inspiration would follow, and the deepest would occur when the largest quantity of venous, and the smallest quantity of arterial, lay in the lungs. The red blood, reaching the left heart, would excite it to those frequent and irregular contractions which accompany the respiratory distress, but frequent and irregular, they would be also ineffectual; red blood would begin again to accumulate in the left heart, the pulmonary veins, and the lungs; till at last their capillaries would contain little else, and the exciting cause of inspiration, the venous blood, being no longer present, the act itself would again cease."

From the preceding quotation, it would seem that Dr. Little's very ingenious theory is based upon the assumption, that to derangement of dynamic adjustment between the right and left ventricle of the heart, is primarily and essentially due the phenomenon of respiratory disturbance. There are, however, certain difficulties in the way of its acceptance, which I shall here content myself with stating. The principal of these Dr. Little has anticipated; namely, that whilst derangement of equilibrium between the two sides of the heart, by enfeeblement, actual or virtual, of the left ventricle, is very common, respiratory distress of this peculiar character is comparatively rare. For example; advanced fatty degeneration of the left ventricle, the right being very slightly, if at all, affected, is not uncommon.

Neither is attenuation of the left ventricle without a corresponding change in the right, in old, ill nourished, and hard worked persons. Yet in neither of these cases, where other and very palpable changes have not taken place, is the phenomenon in question witnessed. Again, lesions of the aortic valves, involving obstruction and inadequacy, and obstruction at the mitral orifice, which is no less effectual in the production of arterial blood-stasis in the lungs, though of frequent occurrence, are rarely associated with this symptom, and never so associated in the absence of other changes.

Finally, the assumption involved in this theory, that the *besoin de respirer* has its seat in the air vesicles of the lungs, and for its cause, the reflex irritation arising from the presence in them of carbonic acid, is unwarranted. The experiments of Flint* have clearly proved that the feeling of the "want of air" has its seat, not in the lungs exclusively, but in the tissues of the body generally; and that the excitant of that feeling is not carbonic acid by its presence, but oxygen by its absence.

I have already stated that the only lesion of structure with which rhythmical irregularity of breathing has been always found associated, is atheromatous or calcareous change, with dilatation, of the arch of the aorta, involving loss of elasticity in its walls. I think these changes supply the conditions of a rational theory of the phenomenon. During the period of greatest tranquillity of the heart's action, viz., in sleep or repose, the systemic capillary circulation fails, from want of the contributory aid rendered in health by the elastic reaction of the aorta. Hence arise a suspension of tissue-respiration, *besoin de respirer*, and accelerated or suspirious breathing, as shown by the experiments of Flint already referred to. Accelerated respiration must strengthen capillary circulation; first, through the lungs, and then through the tissues of the body generally, by quickening the action of the heart, and increasing its force. In proportion as the systemic capillary circulation becomes established, the *besoin de respirer* is less urgent, and respiration gradually subsides, till a period of apnoea arrives. The descent of respiration below the normal standard would seem to arise from its previous excessive activity, and the ex-

* *Physiology of Man*, article "Respiration," 1866.

haustion of the patient. Now, again, comes a period of feeble action of the heart, and failure of capillary circulation, with its consequence of paroxysmal breathing. That imperfect circulation of arterial blood in the respiratory centre contributes in a special manner, and in a great degree, to the production of the respiratory derangement, I have no doubt; but the effect of this is not easily distinguished from that of a want of oxygen in the tissues of the body generally. The *fact* that dilatation and rigidity of the arch of the aorta is, *par excellence*, the lesion with which rhythmical irregularity of breathing is associated, and of which it may, therefore, be regarded as pathognomonic, will be in no degree affected by the estimate formed of the foregoing theory. This theory is propounded only with a view of presenting in the form of a connected and rational series the sub-phenomena constituting this remarkable symptom.

Doctor Little observed that the respiratory distress became gradually less urgent, and finally ceased, at the near approach of death.

I have not observed this in any of my cases. Theoretically, I would have expected it, irrespective of the positive testimony of so competent an observer as Dr. Little. Yet its absence in the cases which have come under my notice deserves to be mentioned, as showing that where the respiratory phenomenon under consideration constitutes a feature of the final illness, it may continue up to the moment of death. Indeed, in a few instances it seemed in no inconsiderable degree to aggravate the last sufferings of the poor patient.

Neither have any of my cases exhibited the marked change in the rate of cardiac pulsation, at the different periods of the paroxysm, which Dr. Little has noticed. I have, on the contrary, as already stated, observed either an absolute uniformity of the pulse throughout the attack, or, as in one or two cases, a slight acceleration during the period of dyspnoea, and a gradual decline in that of descent, till a minimum rate was reached on the accession of apnoea.

In 1864, and again quite recently,* Professor Laycock expressed the opinion that this phenomenon depends upon "a

* *Dublin Journal of Medical Science*, July, 1873.

sentient palsy of the respiratory centre," or "a paresis of reflex sensibility of the mucous membrane of the lung;" that "the slower breathing is due to the diminishing sensibility, and the accelerated breathing following the interval of apnœa is due to the stimulus of unaërated blood being hurried proportionately to the need of oxygen." It is, he thinks, "a neurosis of the vagus, not necessarily dependent on structural or other diseases of the heart," and most frequently occurs during sleep, or at the moment of going to sleep; showing that "the gradually diminished motor activity is coincident with gradually diminished sensory activity, until the carbonized blood rouses up the sensory centre."

That lowered sensibility of the vagus centre is a prominent factor in this phenomenon, I am quite in accord with Professor Laycock in holding; but I cannot agree with him in the opinion that it is not necessarily connected with structural disease of the heart, at least, when the latter is associated with disease of the aorta. It is conceivable that diminished sensibility of the vagus centre should be a not unfrequent consequence of one or more of the various forms of organic disease or injury of the brain. It is certainly an associated condition in coma, to whatever cause due. Yet, rhythmical disturbance of breathing is not a symptom of cerebral lesion, or of the comatose state. Starting from sleep, especially when lying on the left side, and in a state of great alarm and dread of some indefinite evil, are usually associated with rhythmical derangement of respiration. The tongue is generally loaded, the stomach distended with flatus, and the bowels constipated, no doubt owing to congestion of the portal system.

Dropsy is not a proper symptom of fatty degeneration of the heart, although it not unfrequently is associated with that affection; but, primarily so, only when the kidneys and liver are likewise engaged. In the advanced stages it is often present, not from the special tissue-change in the heart, but from weakness and dilatation consecutive to it. It existed, in some form, in 25 out of 95 cases in my tables; or, in somewhat more than one-fourth of the whole; that is, in 5 cases, in Table V.; in 8, in Table VI.; and in 12, in Table VII.*

Doctor Ormerod noted it in 10 of his 25 cases.

* *Vide* p. 648, *et seq.*

I may, however, remark, as a clinical fact of which I do not venture to offer an explanation, that oedema of the lower limbs, and extending to the external genitals, is eminently suggestive of a fatty state of the heart and kidneys conjoined. I have not met with a single instance of it, in which, on dissection, this twofold lesion was not found.

Granular degeneration of the kidneys existed in 7, and fatty degeneration of these organs in 11 instances out of the total of 95 cases in my tables; and in those of Dr. Quain, comprising 83 cases, the numbers were 9 and 2 respectively.

The liver is usually engorged in the advanced stages of fatty degeneration of the heart, owing to weakness and dilatation of the right chambers. There is, then, fulness and tenderness on pressure at the epigastrium, and the liver may be felt descending to a greater or less distance towards the umbilicus. But occasionally the liver is primarily engaged, either by cirrhosis or fatty change. The former was witnessed in 4 instances out of the 95 cases included in my tables; and the latter in 12. Where jaundice occurs, the condition of the liver is not that of simple engorgement; and where, with this symptom present, cirrhosis is excluded, fatty liver may be diagnosed.

Arcus senilis is regarded by Mr. E. Canton as pathognomonic of fatty disease of the heart.* He declares that he has "in no instance found this senile arc, when well developed, unaccompanied by fatty degeneration of the heart; and further, that "the extent of change in the cornea may be regarded as a measure of the degree to which the heart-fibre has, in the same manner, become changed." He adds that Dr. C. J. B. Williams had found the "arcus" in 23 cases out of 25 of fatty heart. Of this number, however, the diagnosis was verified by *post mortem* examination in only 2 instances.

Doctor Fuller regards this sign "as of little value in the diagnosis of fatty degeneration of the heart."† And Doctor Haskins gives a list of 12 cases of "arcus," in only 2 of which were there any symptoms referable to the heart; and in 1 only could any organic alteration be suspected; 6 out of the

* *Lancet*, May 11th, 1850, p. 561; and January 18th, 1851, p. 67.

† *Diseases of the Chest*, 1862.

12 patients were perfectly healthy.* This evidence, though strong, is not conclusive, in the absence of proof by dissection, that fatty change of the heart did not exist. Arcus existed in 8 only of the 95 cases included in my tables; viz., in 6 of those in Table V., and in 1 in Table VI., and Table VII., respectively.†

Doctor Milner Fothergill regards the dim and hazy form of "arcus," with ill defined edges, fading off into a cloudy cornea, as pathognomonic of degenerative changes, and eminently suggestive of a state of fatty change of the heart.‡ Whilst admitting that a cloudy ring in the outer margin of the cornea, positively indicates fatty decay and disintegration of that structure, and suggests similar changes in other tissues and organs, I am not prepared to concede that those tissues must necessarily be muscular, and the organ engaged, the heart.

I cannot, therefore, accept either of Mr. Canton's propositions; namely, that the heart-substance must necessarily be in a state of fatty degeneration where well formed "arcus" exists; and that the latter may be taken as a measure of the former condition.

Where strong and direct evidence of a morbid condition of the substance of the heart existed, I should consider the presence of arcus senilis as warranting the presumption that the tissue-change in progress was fatty degeneration. This condition, when present, is not necessarily universal as regards similar structures, much less as regards those which are histologically dissimilar. The liver alone may be the seat of fatty change; the muscles of one or more of the limbs; or even a particular group of congenious muscles, may alone be found to have undergone fatty degeneration.

It is no less certain that the corneæ may exhibit the fatty arc, without the heart being in a similar state of histological change; and, again, that the heart may be the subject of fatty degeneration when no arcus senilis exists. The "arcus," in short, may be regarded as a presumptive, but not as a positive symptom of fatty degeneration of the heart.

The *physical signs* are individually inconclusive, but collec-

* *American Journal of the Medical Sciences*, January, 1853.

† *Vide*, p. 648, *et seq*

‡ *The Heart and its Diseases*, 1872, p. 170.

tively they are of positive significance. Of these, the most important have reference to the sounds of the heart. The first sound is always short and faint. In the early stages it is likewise dull and ill pronounced, the impulse element, though feeble, masking the valvular in different degrees. When fatty degeneration is secondary to hypertrophy, the valve-click is completely muffled at the apex; but when, on the other hand, the tissue-change is primary, the walls of the heart being of normal or reduced thickness, the valve-element of the first sound is faintly audible.

In the advanced stages of primary fatty degeneration, the first sound so closely resembles the second, that, in the absence of cardiac impulse, it becomes necessary to place the finger on the carotid in order to determine its rhythm. This is manifestly the result of a twofold cause; namely, the weakness of the heart, by which the impulse-element is suppressed, and the attenuation of its walls, through which the valve-sound is conducted more distinctly to the ear. Where attenuation has not existed at any stage of the affection, as in hypertrophy undergoing the fatty change, the first sound is dull at the apex, and sharp and valvular at the base, throughout.

Out of the total of 95 cases in my tables, the first sound was dull in 16; i.e., 5 out of 55 in Table V.; 7 out of 18 in Table VI.; and 4 out of 22 in Table VII. The second sound was sharp and clear in 7 instances; viz., 2 in Table V., 1 in Table VI., and 4 in Table VII. I have, in a few instances, found mitral regurgitant murmur of a soft and blowing quality, and adynamic origin, in connexion with fatty degeneration and softening of the left ventricle; the valves, on dissection, were discovered to be perfectly sound and competent. This I am obliged to attribute to yielding of the parietal attachment of the papillary muscles at the acme of ventricular systole.

From dilatation with thinning of the walls of the left ventricle, without structural change, as exemplified in middle aged, hard worked, ill fed and fretful subjects, chiefly females, fatty degeneration with parietal thinning, may, in most instances, be readily distinguished by the physical signs alone, and even where the distinction is not practicable by reference to the history and general symptoms.

In dilatation with thinning, the sounds are sharp, ringing, of normal length, remarkably loud, and extensively transmitted over the chest both before and behind. A distinct apex-pulsation is detectable by the hand to the left of the ordinary position, characterized by a swell of gradual elevation and subsidence, but without tension. The pulse is weak and regular, varying in rate under slight excitement or emotion, and not unfrequently intermitting at distant intervals. The patient is anæmic, and, with the exception of dyspnoea consecutive to intercurrent pulmonary congestion, to which the patient is liable on slight exposure, and this of the ordinary character, there are present none of the symptoms which specially belong to fatty disease of the heart.

In the latter disease, the first sound at the apex is, on the contrary, either masked or sharp according to the thickness of the walls of the left ventricle; but it is always brief and faint, and not audible beyond the precordium, except feebly in the course of the ascending aorta, and precordial impulse of any kind is rarely perceptible.* It is unnecessary to specify the other symptoms already discussed, as characteristic of fatty degeneration of the heart; viz., irregularity or slowness of pulse, pseudo-apoplectic seizures, syncope, rhythmical irregularity of breathing, and arcus senilis.

Doctor Fuller deems it impossible during life to distinguish between simple softening, fatty softening, and attenuated dilatation.† I cannot admit simple softening, as distinct from typhoid or fatty softening. Typhoid softening is readily distinguished by the rapid development of its proper signs, and by the coexistence of fever of the typhoid type. Attenuated dilatation may, I think, be readily diagnosed by the rules just stated.

The diagnosis of fatty degeneration consecutive to hypertrophy, and especially the determination of the period when this change is inaugurated, are of much consequence; because of the extreme gravity of this form of the affection, and the advantage

* Doctor Stokes remarks that occasionally, in cases of extreme fatty degeneration of the heart, a diffused impulse may be felt without apex-beat, resembling the heaving of a thin-walled aneurism. *The Diseases of the Heart and Aorta*, p. 328.

† *Opus citat.*

of an early application of hygienic and therapeutic measures, with a view to the arrest of tissue-decay.

The characteristic symptoms of this change are implied in the expression, *failure of the heart*. Thus, one of the earliest symptoms, indeed in one class of cases the earliest, is tumultuous action or palpitation, with feeble and irregular pulse, when the patient makes any sudden effort ; it is most likely to occur if the patient rapidly ascend a steep incline, such as a flight of stairs, or hurry over a journey on foot, being urged forward by a desire to be up to time. Such is usually the first indication of "break up" in those who at the middle period of life, have enjoyed good health, with the exception of occasional twinges of gout, and have been of active habits, and lived freely ; or, if of the working classes, who have indulged in the use of malt drink. An eminent medical practitioner in a country district, of middle age and full habit, informed me that the first intimation which he received of disease of his heart, consisted in palpitation, precordial oppression, and overpowering dyspnoea, on rapidly ascending a flight of stairs in his own house, in order to procure some medicine before answering a hurried call. He was ever afterwards subject to paroxysms of dyspnoea, which gradually became more frequent, and would ultimately attack him even when at rest. I attended this gentleman in his last illness, which was characterized by all the symptoms and signs of enlarged and fatty heart, with atheromatous and dilated aorta. (See Case 47, J. N. W.)

The case (51), previously referred to (p. 627), affords another example of the same kind. A working mechanic, over sixty years of age, who had lived well, but temperately, noticed for some time slight shortness of breath on exertion. He received a severe shock from the fall of a beam of wood upon him, and was thenceforward subject to more severe accessions of dyspnoea. A week previous to my seeing him, he was proceeding hurriedly to his work after his morning meal, when he was suddenly seized with dyspnoea and oppression, so urgent that he was forced to return home, and was never afterwards able to resume his labour. There was present all the evidence of enlarged and fatty heart, dilated aorta, and disease of the mitral and aortic valves, which was subsequently confirmed by dissection.

The history of all such cases is that of atheromatous aorta, with or without valvular disease and hypertrophy, and, subsequently, fatty degeneration and dilatation of the left ventricle.

The two last mentioned changes are those which determine the symptoms of failure of the heart. When fatty degeneration supervenes upon hypertrophy from valvular disease at the aortic or mitral orifice, although the "break up" of the general health is more or less abrupt, and usually attributed to a definite cause involving muscular effort, still, as in the preceding example, the surprise is not complete; because, from the previous existence of symptoms referable to the valvular lesion, the patient must have been sensible that he was the subject of some form of cardiac affection. Here likewise, however, failure of the heart from fatty degeneration and dilatation is the immediate cause of the critical change. When symptoms of this critical character have been exhibited, it is too late to attempt anything with a view to arresting the progress of the disease. But if the patient had been previously under skilled observation, it would be the duty, as no doubt it would be within the competence of a well informed practitioner, to determine from the occasional presence of vertigo or amblyopia, irregularity of pulse, and slight feeling of suffocation with hurried breathing, the incipience of fatty degeneration of an hypertrophied heart. Then it is, that by regulating the diet and habits of the patient, and putting him promptly under a suitable course of medicine, the process of retrogressive change may be retarded, or even arrested.

In other and not fewer instances, the pathological series commences with granular degeneration of the kidneys, accompanied not unfrequently by atheromatous change in the coats of the aorta. Then follow in succession hypertrophy, fatty degeneration and dilatation of the left, and next, of the right ventricle, and fatty conversion of the kidneys themselves. In this latter case, cedema of the feet is usually the first, as effusion into the serous cavities constitutes the last, member of the series. These are usually the most urgent and distressing cases; they are not unfrequently complicated with erysipelalous inflammation and sloughing of the lower limbs. The presence of albuminuria, with a low specific gravity of the urine, would here constitute the first symptom of

disease ; but, as in the former case, the earliest indications of fatty change in the heart would have reference to its failure. Where fatty degeneration is secondary to inflammation of the heart, and not preceded by hypertrophy, the substance of the heart has been the seat of the inflammatory process. In other words, of the phlegmasiæ of the heart, myocarditis alone is capable of leading directly to fatty transformation of its substance. This latter is the most rapidly fatal process of fatty change to which the heart is liable, because of the absence of the protective influence of hypertrophy.

The heart was hypertrophied in 39 out of a total of 68 cases of fatty degeneration reported by Quain. It was normal in 8, and atrophied in 4 instances. In my list of 95 cases, the heart was hypertrophied in 31 instances, and dilated in 37.

Doctor Quain inquires whether hypertrophy may not proceed "from a natural effort to compensate for loss of power." I think not ; because the antecedent state of fatty degeneration implies impairment of nutrition.

A certain incompatibility has been assumed to exist between fatty degeneration of the heart and valvular disease.

Doctor Henry Kennedy declares that out of a list of 245 cases of fatty heart, collected by him from different sources, there were only 33 in which disease of the valves existed.* He refers to a list of fifty deaths from the administration of chloroform, published by Snow, in nearly all of which fatty disease of the heart *without* valvular lesion was found ;† and adds, that when valvular lesion exists in such connexion, it is usually located at the orifice of the aorta, the valves being not atheromatous but fatty and competent.

Doctor Walshe mentions "absence of murmur" as negative evidence of fatty heart.‡

Doctor Stokes, on the contrary, is of opinion that "valvular disease is a not infrequent combination with fatty heart." In most of the cases which he has seen, the valvular affection was at the aortic orifice ; and he calls attention to the existence of

* *Proceedings of Surgical Society of Ireland*, March, 1864.

† *Proceedings of the Pathological Society of Dublin*, vol. i., January 8th, 1859.

‡ *The Diseases of the Heart*, third edition, 1862, p. 351.

aortic systolic murmur, either blowing or musical, accompanying or replacing the first sound, the second sound being either normal or suppressed, but *without* the signs of regurgitation, as confirmatory evidence of fatty heart. He admits only two forms of valvular lesion in connexion with a fatty state of the heart; viz., those causing aortic, and mitral obstruction.*

Jaccoud tries to establish a special connexion between aortic valve-inadequacy and fatty degeneration of the heart; urging that, inasmuch as the coronary arteries receive their charge of arterial blood during diastole, regurgitation at the aortic orifice must diminish both the volume and the pressure of the coronary circulation, and must consequently lower the nutrition of the heart. He maintains, however, that the degenerative change is of a mixed or "fibro-fatty" character, relying upon the authority of Billroth and Traube, who assert that muscle tends to degenerate into connective tissue whenever its nutrition is impaired. He thinks it probable that, in such cases, myocarditis progressed concurrently with the endocarditis which disorganized the valves, and favoured the occurrence of tissue-degeneration.† With regard to this statement, I may observe that I have not investigated the matter specially in relation to aortic reflux; but in ordinary fatty degeneration, even that depending manifestly upon defective supply of arterial blood, I am in a position positively to deny the existence of any increase of the plastic or fibroid element. In reference to the assumed origin of the valvular lesion from endocarditis in such cases, and the argument founded thereon, I will only remark that, in my opinion, this lesion is rarely of inflammatory origin, and is most frequently of a degenerative type, like the fatty change in the heart itself, and is coeval with it. A "disorder of compensation," such as hypertrophy of the left ventricle consequent upon aortic insufficiency, may be converted into "derangement of compensation." When the latter is due to a temporary cause, such as fatigue, or bronchitis, it is not of grave augury; but when it results from the natural progress of the disease, as exemplified in mitral incompetence from fatty softening and dilatation of the

* *The Diseases of the Heart and Aorta*, 1854. p. 330-1.

† *Medical Press and Circular*, July, 1868, translated by G. Cockle, M.D.

left ventricle, consecutive to hypertrophy from incompetence of the aortic valve, then the prognosis is very grave, and a state of permanent asystolie is established. On this subject, Tables V., VI., and VII. (pp. 648, 656, and 660), show the following results :

Condition of Valves.	Table V. 55 Cases.		Table VI. 18 Cases.		Table VII. 22 Cases.		Total. 95 Cases.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
Valves normal ..	16	28.80	12	66.60	9	40.90	37	38.95
Aortic valve diseases ..	9	16.36	1	5.50	8	36.36	18	18.90
Mitral valve diseases ..	6	0.90	9	40.90	15	15.80
Not stated ..	21	38.18	5	27.70	26	27.36
Aortic-systolic murmur	4	7.27	8	36.36	12	12.60
Aortic-diastolic murmur	1	1.81	1	5.50	2	9.09	4	4.20
Double aortic murmur	3	5.45	2	9.09	5	5.26
Mitral-systolic murmur	1	1.81	3	16.60	7	31.81	11	11.60
Left presystolic murmur	1	1.81	1(?)	4.54	2	2.10

Thus it would appear that, if the instances, 26 in number, in which the state of the valves is not mentioned, be deducted from the total of 95 cases, the residue is nearly equally divided into those in which the valves were unaffected, and those in which one or more sets of them were disorganized; the proportion being as 37:33.

In 34 instances, murmur of some kind was present; and of these it is noteworthy that it was located at the aortic orifice in no less than 21 instances, being systolic in rhythm in 12 of the number; and at the mitral orifice in 13 instances, being likewise systolic in 11 examples.

In Table VI., for the items in which I am personally accountable, aortic murmur is stated to have existed in 12 instances, whereas disease of the aortic valve was actually present in only 8. In the remaining 4 cases, the murmur was produced in the aorta itself by atheromatous or calcareous roughening of its inner surface.

The practicability of diagnosing a fatty condition of the heart, especially in its early stages, has been questioned by some eminent authors. Dr. Latham denies it, and Dr. Stokes expresses a

qualified opinion in the following terms: "The fatty degeneration considered alone, does not, so far as we know, afford any special or separate sign; and it would be difficult or impossible to draw a line of distinction between the signs of simply weakened heart, and of this condition combined with fatty degeneration." "In many minor cases of this condition, the heart's sounds may present no abnormal character."*

It will be observed that the preceding opinion has reference to the signs exclusively. I think, however, that the absence of the impulse, or its extremely feeble character; the brief duration of the first sound, whether masked or sharp, in primary cases, and its almost complete or absolute extinction in those preceded by hypertrophy; the restriction of the sounds within a very limited area; and the occasional irregularity of the heart's action, will suffice, in the majority of cases, to establish the diagnosis of fatty heart from the physical signs alone.

The existence of a single murmur of systolic rhythm at the root, or in the ascending portion of the arch of the aorta, may be taken as strong collateral evidence of a fatty state of the heart; because of the very frequent association of this condition with atheroma of the aorta, involving the valves or not. The aorta was atheromatous in 40 out of 95 cases epitomized in my tables; nor should this be matter for surprise, atheromatous transformation of the aorta being itself a veritable fatty degeneration, as shown by Gulliver and Virchow. The absence of diastolic basic murmur affords, I think, in such cases, no proof of the competency of the aortic valves; the reaction of the aorta, owing to the loss of its elasticity from disease of its coats, being inadequate to the production of a reflux current of sufficient force to develop a murmur. If, at the same time, independent evidence of hypertrophy, with dilatation and elongation of the left ventricle, be present, as afforded by downward and outward displacement of the apex, collapsing, and visible pulse, and throbbing of the carotids, the existence of aortic regurgitation may be confidently affirmed, notwithstanding the absence of the characteristic diastolic murmur.

If the typical symptoms of occasional vertigo, momentary

* *Opus citat.*, p. 327-8.

failure of sight, dizziness, syncope, or pseudo-apoplexy, and paroxysmal and rhythmical dyspnoea* coincide in any given case with the physical signs just mentioned, the positive diagnosis of fatty heart would be fully warranted.

The incipience of primary fatty degeneration may be suspected, if the pulse, previously regular, become weak and irregular; if the surface be pale, the patient subject to dizziness or syncope, and the cardiac impulse feeble; although the sounds of the heart may not appreciably differ from their normal character. A positive diagnosis, however, would be impracticable under the circumstances.

The mode of death is most frequently slow, and by a gradual process of sinking or exhaustion. Less frequently it takes place by a gradual process of a different kind; namely, by asphyxia, or by coma. It may likewise occur, rapidly, from angina pectoris, from a leakage of blood into the pericardium, or from rupture of the septum ventriculorum; or, instantaneously, by simple syncope, or from rupture to a large extent of the outer wall of the heart, or of the aorta. Amongst Ormerod's and Quain's cases of fatty degeneration, 93 in number, death occurred by syncope in 10, by coma in 1, and by rupture of the heart in 22 instances. The following is a summary, in tabular form, of the mode of death in 83 out of the 95 cases comprised in Tables V., VI., and VII.:

MODE OF DEATH.

Causes.	Table V. 55 Cases.		Table VI. 18 Cases.		Table VII. 23 Cases.		Total. 95 Cases.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
Asthenia	23	41·81	5	27·70	17	77·27	45	47·37
Angina Pectoris ..	3	5·45	1	5·50	4	4·21
Asphyxia	1	1·81	4	22·20	2	9·09	7	7·37
Coma	3	5·45	1	5·50	3	13·63	7	7·37
Rupture of heart ..	15	27·27	2	11·10	17	17·89
Rupture of aorta ..	6	10·90	1	5·50	7	7·37

With regard to the seat of rupture, in these 17 instances; it occurred on the anterior wall of the left ventricle in 8 cases; on the posterior wall of the left ventricle in 6; on the anterior sur-

* The significance of this symptom depends upon the very common association of fatty heart with dilated and atheromatous aorta, of which it is pathognomonic.

face of the right ventricle in 1 ; through the septum ventriculorum, not communicating with the exterior, in 1 ; and through the septum ventriculorum, ultimately penetrating the anterior wall of the right ventricle, in 1 instance. In the last mentioned case the patient lived six days after the occurrence of the primary rupture.

It will thus be seen that the outer wall of the left ventricle was the seat of rupture in no less than 14 out of 17 examples of this accident, and that it occurred through the anterior, more frequently than through the posterior wall, in the proportion of 8 : 6.

Of Quain's 22 cases, rupture of the external wall of the left ventricle took place in 17 ; viz., on the anterior surface of the ventricle in 12, and on the posterior surface in 5 instances. The septum ventriculorum was rent in 1 instance, the right ventricle in 2, and the right auricle in 2 cases.

It is noteworthy, that when the septum ventriculorum was the seat of rupture, the patients invariably survived the accident several days. Such was likewise the case in the examples, five in number, of partial rupture of the wall of the heart. One such accident occurred through the deep layers of the left ventricular wall ; one through those of the right ventricle ; one in a similar situation in the right auricle ; and one through the superficial fibres of this chamber, not communicating with its interior.

The symptoms indicative of rupture of the heart, with the exception of that of the septum of the ventricles, are sudden and severe lancinating pain, accompanied with a feeling of sinking and fluttering in the region of the heart, arrest of respiration followed by a few gasping inspiratory efforts at long intervals, failure of the pulse, extreme pallor quickly succeeded by capillary venous engorgement of the cutaneous surface, loss of consciousness, and death within a period of a few minutes. When the septum ventriculorum alone has been rent, the patient usually survives several days ; and in one instance, already mentioned, a period of six days actually intervened between the date of this accident and that of death. Dr. Quain mentions a case in which the patient survived eight days.*

* Lumleian Lectures, *Lancet*, March, 1872.

Three of the most eminent Scotchmen of the last generation, Drs. Chalmers, Abercrombie, and Sir J. Y. Simpson, died of fatty disease of the heart; the two former suddenly. Dr. Chalmers died of simple syncope, and Doctor Abercrombie from rupture of the left ventricle. In Dr. Chalmers' case there were no premonitory symptoms, except an attack of temporary hemiplegia, from which he entirely recovered in the course of a few weeks, thirteen years before death. Dr. Abercrombie had, four years before death, an attack of faintness, with oppression and headache of brief duration, but no other premonitory symptom. The heart was in a state of fatty degeneration. Dr. H. Bennett reported on Dr. Chalmers' heart, the left ventricle of which was almost entirely converted into fat, exhibiting under the microscope lines of granules, and no transverse striation.* Sir J. Y. Simpson died of fatty degeneration of an hypertrophied heart, but by gradual exhaustion, or asthenia.

The following Tables, V., VI., and VII., have been constructed from the records of the London and Dublin Pathological Societies, and from my own register, respectively. They exhibit, in summary, the latest available contributions, in a clinical aspect, to the pathology of fatty disease of the heart; and, commencing from the date of publication of Dr. Quain's valuable statistics (May, 1850), they may be regarded as supplementary to his Tables, and as representing the present state of clinical knowledge of the subject in the United Kingdom.

* *Contributions to Practical Medicine*, by James Begbie, 1862.

TABLE V.—FATTY HEART.
Pathological Society of London (from May, 1850).

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Bagshawe	M.	..	Tailor	Many years	Palpitation, faintings, pain in chest	..	Syncope by rupture of aneurism of fat of left ventricle	Atheromatous	Slightly atheromatous	Heart thinned; layer of fat on surface; rupture of aneurism of left v.	Fatty degeneration; (slight)	Coronary arteries calcified, especially branch supplying seat of aneurism; kidneys granular.
Dr. Daly	F.	52	A lady	3 years	Temporary right hemiplegia, headache, "black dimittance," weak and intermittent pulse	Weak action and sounds of heart	Rupture of left v. on posterior surface	Atheromatous and calcified	Mitral slightly thickened; aortic also thick and calcified at root	Left v. hypertrophied and dilated; much fat at base and in solid	Fatty degeneration; greater in seat of rupture	Old blood-clot in brain, and degeneration of arteries at its base; degeneration of coronary arteries, and advanced degeneration and closure of branch leading to seat of degeneration.
Dr. Barker	M.	45	Mariner	A few weeks	Dyspnoea and palpitation; pulse 120, moderately full, & jerking; drowsy	Extensive precordial dulness, yellowish, pulse to be felt, and sounds very faint	Died suddenly after excitement (syncope?)	Atheromatous	Aortic much diseased	Left v. $\frac{1}{2}$ in. thick; right v. dilated; heart 28 oz.	Early fatty degeneration	Aneurism of abdominal aorta, and granular degeneration of kidneys; degeneration of arteries of brain, and local softening of latter.
Dr. Barlow	F.	29	"Irregular"	Coma from injury of head	Slightly atheromatous	Fatty degeneration (general)	Brain reduced in volume, tough, and in the state of "fibrous degeneration" of sinus and Crivellier; coronary arteries atheromatous; kidney's small and cystic; arcus semilis; emphysema of right lung.
"	M.	—	Cabman	Asthenia	Dilated and atheromatous	..	Dilated generally	Granular degeneration; more extensive in left v.	Coronary artery leading to rupture calcified and plugged.
Dr. Belth	M.	79	..	Some years	Dyspnoea, vertigo, faintings, pain in chest	..	Rupture through septum of v. extending through anterior wall of right v.; both v. covered with fat to depth of one-half; lived six days after signs of rupture of septum	Healthy	Healthy	Heart hypertrophied, 17 oz.	Fatty degeneration, early stage	

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibra.	State of other Organs.
Dr. Bristowe	M.	35	..	5 months	Cough, debility, weak and intermittent pulse	Those of pleuro-pneumonia engaging lower half of left lung	Asthenia	Healthy	Healthy	Recent pericarditis with effusion; no adhesion; heart covered with shreds of lymph, and substance yellow, to depth of half of right v., and one line of left v.	Yellow portion in state of fatty degeneration; retraction; the former shading into the latter part, which was <i>very brittle</i>	Left pleuro-pneumonia, and right ditto in less degree; liver congested.
"	M.	68	Embarrassed and desponding	..	Rupture of left v. posteriorly	Atheromatous	Mitral and aortic slightly atheromatous, but competent	Hypertrophied (16 oz.); right v. loaded with fat	Slight fatty degeneration generally, but advanced in seat of rupture	Coronary arteries atheromatous; not more so in seat of rupture than elsewhere.
"	M.	11	.	6 weeks	Depression, general purpuric mottling, hemorrhages; finally, loss of vision; dilated pupils, and "muscae;" pulse large, and quick	..	Asthenia	Healthy	Healthy	Normal as to size; mottled; yellow on interior of left v.	Advanced fatty degeneration in seat of rupture	Brain remarkably white and anemic, otherwise healthy.
"	M.	26	Clerk	6 months	Jaundice, vertigo, vomiting, debility; pulse quick, and weak	Action and sounds of heart reported normal	Asthenia	..	Healthy	Both v. hypertrophied, but not dilated; weight 13½ oz.	Fatty degeneration of internal layer of both v.	Liver fatty.
"	M.	10	..	1 month	Diphtheria	..	Asthenia	Heart small and contracted	Fatty degeneration, especially in patches on inner surface of right v.	Kidneys fatty.
Mr. Canton	M.	49	Gentleman	Syncope	Left v. hypertrophied, except at apex, where it was thinned, so as to consist of endo- and pericardium only; this portion was of the size of a pigeon's egg; general adhesion of pericardium	Fatty degeneration advanced in vicinity of aneurysm only	

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Walls of Heart.	State of Fibra.	State of other Organs.
Mr. Canton ..	M.	72	Spring-maker	3 years	Dyspnoea, and occasional pain in chest	..	Rupture of aneurism of aorta with in pericard.	Atheromatous	Healthy	Large and flabby	Fatty degeneration	Coronary arteries atheromatous and calcified.
Dr. Chlodelmely	M.	48	County baronet	5 years	Attacks of angina pectoris; gouty.	..	Cardiac syncope	Atheromatous (and also pulmonary artery)	Healthy (slight thickening at base)	Ventricles slightly dilated and streaked yellow on both surfaces	Fatty degeneration	Coronary arteries calcified; arcus senilis.
Dr. J. Cockle	M.	40	Soldier (ex.)	2 years	Pains in chest and arms occasionally; inability to lie on left side; cough	Dull first sound at apex, <i>sharp</i> second at base; then a double pulsating tumor to right of sternum, with double sound; first sound in this situation became "crumpling" shortly before death	Rupture of bilocular aneurism of aorta into pericardium	Dilated and atheromatous	Healthy	Left v. hypertrophied	Fatty degeneration	Adherent pericardium.
Mr. Coulson	F.	76	Rupture of left v. behind apex	Covered with fat to one line from inferior	Fatty degeneration, (advanced)	Coronary arteries calcified.
"	M.	46	None preceding the syncope, of half an hour's duration, in which he died.	..	Syncope by rupture of aneurism of aorta into pericardium	Atheromatous	..	Loaded with fat	Fatty degeneration	
Dr. Crisp ..	M.	39	School-master	10 months	Pain in precordium extremely acute and continual for the last few weeks of life	..	Syncope	Atheromatous and contracted	Aortic valves shrunk and contracted	Both v. dilated and thickened	Fatty degeneration	
Dr. T. W. Davis	F.	64	Nurse	8 months	General dropsy, dyspnoea, weak and irregular pulse; angina pectoris	Extensive precordial dulness; tumultuous action of heart; double basic murmur	Rupture of right v. anteriorly	Atheromatous	Aortic much disorganized	Right v. thinned & ruptured in front; left v. dilated and thick	Fatty degeneration	Coronary arteries calcified, especially right
Dr. Dickinson	M.	36	..	Few months	Antecedent peritonitis, and acute pains in feet	..	Exhaustion	Slightly atheromatous	..	Large and flabby	Fatty degeneration	Adhesion in pericardium, and coagulation of blood in arteries of lower limbs.

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Pulsa.	State of other Organs.
Dr. Dickinson	M.	44	Butler	2 years	Extreme anemia and emaciation from want	Double murmur at lower sternum	Exhaustion	..	Mitral and tricuspid rough	Heart large and flabby, with superficial layers of fat; fawn-coloured and mottled yellow	Extreme fatty degeneration	All organs anemic; blood watery.
Dr. Fuller	F.	40	..	7 months	General malaise after tapping an ovarian cyst 4 years previously; jaundice and colic of feet	..	Progressive asthenia	..	Normal	Apparently normal	Fatty degeneration (advanced)	Tubercle in apex of right lung, and collapse of adjacent part of it.
Dr. G. Gibb.	F.	45	Apoplexy	Dilated and atheromatous	Healthy	Left v. hypertrophied	Fatty degeneration of left v.	Liver fatty; arteries of brain atheromatous, and clot of blood projecting into right lateral ventricle.
"	M.	60	Those of phthisis	..	Asthenia	Atheromatous and calcareous	..	Heart normal in size, but soft	Fatty degeneration (advanced)	General calcification of arteries, including coronary; fatty liver and kidneys; tubercle and cavities in lungs; larynx and trachea calcified.
"	M.	46	Cerebral hemorrhage	Atheromatous	..	Heart large	Fatty degeneration	Larynx and trachea calcified.
Dr. Greenhow	M.	65	Painter	8 years	Cough, and shortness of breath	Systolic murmur at lower sternum; extended precordial dullness, and weak impulse	Assthenia	Atheromatous	None	Heart large (25 oz.), and generally dilated	Fatty degeneration	Emphysema of lungs, and some cheesy tubercle.
Dr. Habershon	F.	23	"Disolute"	..	Dyspnoea; articular rheumatism	Aortic-diastolic murmur; action of heart tumultuous	..	Asphyxia	Aortic valve disorganized	Heart slightly enlarged; small aneurism in wall of left ventricle	Fatty degeneration, in patches and streaks	Kidneys cystic; double pleuritis and pericarditis with effusion.
Dr. G. Harley	F.	65	..	A fortnight	Cough and slight dyspnoea	..	Rupture of left v. coronary artery	Normal as to size, but soft, pale, and flabby	Fatty degeneration	..
"	F.	28	Householder	6 months	Spasmodic dyspnoea, weakness, and tendency to faint	..	Syncope	Heart large, pale, flabby, and covered with yellow	Fatty degeneration (advanced)	All organs anemic.
Dr. Hillier	M.	5	..	3 years	Enlarged lymphatic glands and spleen; convulsions before death	..	Exhaustion	Soft, pale, and mottled yellow	Fatty degeneration (extreme)	Liver fatty; spleen enlarged.
Mr. Holmes	M.	60	Suffering from stricture and stones; pulse weak and failing	..	Syncope	..	Healthy	Heart 14½ oz., pale and flabby	Fatty degeneration	..

TABLE V.—Continued.

Author.	Sex	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Handfield Jones	M.	62	..	None preceding final seizure	..	Syncope, after a journey of 86 miles	..	Mitral inadequate	Heart enlarged, 2½ oz.; left v. hypertrophied	General fatty degeneration	Ulcers of inner surface of stomach, regarded as result of fatty change.
Mr. A. Leggatt	M.	18	An invalid from childhood	General paralysis	Sounds and impulse of heart reported healthy shortly before death	Right pneumonia	Normal	Fatty degeneration (incipient)	Fatty degeneration of voluntary muscles; softening of spinal cord; right basic pneumonia.
Dr. Leared ..	F.	11	6 months	General anæmia, dyspnoea; vomiting; quick and weak pulse	A murmur with the first sound	Asthænia	..	Healthy	Somewhat large, pale, and flabby	Fatty degeneration	Pulmonary emphysema (slight), with hydro-thorax.
Dr. Lichtenberg	M.	29	Some time	Palpitation and dyspnoea	Double murmur at base	Rupture of aneurism of aorta into right ventricle	Atheromatous	Aortic valve thick and soft	Heart large, thin, and flabby; also yellow (superficial fat?); aneurism, size of marble, projected into tricuspid orifice, and burst	Fatty degeneration	Liver and kidneys fatty.
Dr. J. W. Ogle	M.	69	..	Had two fits of apoplexy previously	..	Apoplexy	Atheromatous	Fatty degeneration, (early stage)	Atheroma of coronary and of cerebral arteries, with softening of brain and sanguineous effusion.
"	M.	54	..	Pain in back; dysphagia	..	Hæmoptysis by rupture of aneurism into cavity formed by pneumonia in base of right lung	Atheromatous	Healthy, but mitral orifice dilated	Loaded with fat; large and thinned	Fatty degeneration	Base of right lung excavated and communicating with aneurism of inferior thoracic aorta.
"	M.	Mid. age	..	Frequent fits, presumably syncope	..	Fit after full meal	Normal	Fatty degeneration	Aneurism of left middle cerebral artery which had burst into left lateral v.; pleuritic adhesion, and granular kidneys; anous scellæ.
"	M.	39	7 months	Ascites; slight jaundice	Basic-systolic murmur	Asthænia	..	Aortic valve ulcerated	Large and flabby	Fatty degeneration	Liver cirrhotic and fatty; kidneys fatty; spleen enlarged.
Mr. J. Part ..	F.	73	..	Those of enlarged liver and spleen	..	Exhaustion	Large and fatty	Fatty degeneration	Spleen greatly enlarged and fibrous; liver very fatty

enlarged and fatty.

TABLE V.—Continued.

Author.	Sex	Age	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Modes of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Peacock	M.	62	Gentleman	4 days	None preceeding final illness	..	Rupture of septum v.	..	Aortic and mitral opaque and orifice of aorta reduced	Right v. covered with fat; left v. thick; heart enlarged (14½ oz.)	Fatty degeneration (general) greater in seat of rupture	Coronary arteries atheromatous and calcareous.
Dr. Pollock	F.	75	Pain in head for a fortnight before death	..	Found dead in bed, apparently from angina pectoris	Fatty degeneration	Arteries at base of brain calcified.
Dr. Quain	F.	59	A lady	7 years	Three attacks of apoplexy and several of angina pectoris	Weak dull, and prolonged first sound; sharp second sound	Angina pectoris	..	Healthy	Heart hypertrophied (14 oz.); right a. dilated and thick; right v. thick	Fatty degeneration & growth; former part of septum and anterior wall of left v.	Coronary artery leading to degenerated part, calcified.
"	F.	68	A lady	2 years	Dyspnoea, oppression; numbness in left fingers and irregular pulse two days before death; fits of angina pectoris	..	Rupture of left v. on anterior surface	Local fatty degeneration in seat of rupture mixed with fibrous do.	Fatty liver, and calcification of coronary artery leading to degenerated part of left v.
"	F.	66	A cook	Some time	Shortness of breath on ascending heights, and slight pain in chest; weak, irregular, and intermittent pulse	Weak action and sounds, especially second	Rupture of left v. anteriorly	..	Healthy	Right v. dilated, thinned, and loaded with fat; left v. dilated only	Fatty degeneration, and greater in seat of rupture	Coronary artery leading to seat of rupture calcified and obstructed.
"	P.	29	Prostitute	..	Phagedenic ulcer of pudenda	..	Assthenia or "cardiac syncope" under chloroform	Healthy	Healthy	Dilated, flabby, and covered with fat	Fatty degeneration	All healthy.
"	P.	Agel	Cough and dyspnoea	..	Rupture of left v. anteriorly	Dilated, flabby, and covered with fat	Fatty degeneration of left v. greater near seat of rupture	Coronary artery leading to seat of rupture calcified and blocked.

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Quain ..	M.	68	Farmer	2 years	Attacks of syncope	..	Rupture of left v. anteriorly	..	Healthy	Heart slightly enlarged	Fatty degeneration; greater in seat of rupture	Coronary artery leading to seat of rupture calcified.
"	M.	73	Gentleman	8 years (?)	Left hemiplegia; Shortness of breath on making ascent; pulse regular; pain in chest nine days before death	..	Rupture of left v. anteriorly	Loaded with fat, and thinned	Fatty degeneration	Coronary arteries atheromatous.
Mr. R. R. Robinson	M.	43	Carpenter	6 months	Dyspnoea, faintness, and occasional palpitation; very weak pulse; enlarged liver; albuminuria; and latterly, orthopnoea; oedema, and gangrene of legs, and respirations only nine in the minute	Rapid and weak action of heart	Asthenia	Dilated and atheromatous	Aortic diseased	Both v. dilated and thick; pericardium adherent, and calcareous plate in false membrane; congenital opening in pericardium, through which left a. appendix was visible	Fatty degeneration	Liver "nutmeg" and fatty; kidneys fatty.
Dr. J. S. Ramskill	F.	45	A lady	Some time	General symptoms of fatty heart	..	Rupture of left v. anteriorly	Atheromatous	Aortic atheromatous at base	Heart small, soft, and covered with fat	Fatty degeneration	
Dr. Thudichum	F.	33	..	8 years	Uterine and intestinal derangement	..	Right pleuro-pneumonia	Atheromatous	Aortic cribriform	Both v. dilated and thinned	Fatty degeneration and "green pigmentary" ditto (?)	Right pleuro-pneumonia; coronary arteries calcified.
Doctor Ogier Ward	F.	10	..	6 years	Chorea, pallor, lividity and dyspnoea; feeble, quick, and irregular pulse	Dulness and riles over bases of both lungs; extension of precordial dullness; murmur with first sound (presystolic?)	Asthenia	..	Endocardium of left a. opaque, and auricular surface of mitral valve "beaded"; orifice narrowed (?)	..	Fatty degeneration (slight)	
Dr. Wilks ..	M.	61	Coal dealer	8 months	Attacks of angina pectoris	Sounds muffled	Fat of angina pectoris	..	Healthy	Left v. dilated; heart loaded with fat	Fatty degeneration	Coronary arteries calcified.
"	M.	61	Lunatic	Rupture of left v. anteriorly	Normal, but covered with fat nearly to entire depth	Fatty degeneration at seat of rupture (advanced)	Coronary arteries calcified, and branch leading to rupture plugged.

SYNOPSIS OF TABLE V.

SEX :		Cases.
Males		35
Females		20
		—55
AGES :		
Under twenty years		6
Twenty to thirty years		6
Thirty to forty		5
Forty to fifty		11
Fifty to sixty		4
Sixty to seventy		13
Seventy to eighty		6
Not stated		2
"Middle aged"		1
"Aged"		1
		—55
DURATION OF ILLNESS :		
Minimum	four days	
Maximum	eight years	
SYMPTOMS :		
Faintness, intermittent and irregular pulse		15
Paroxysmal dyspnoea		16
Angina pectoris		5
Dropsy		5
Arcus senilis		6(?)
Not stated		8
		—55
PHYSICAL SIGNS :		
Aortic-systolic murmur		4
Aortic-diastolic murmur		1
Double aortic murmur		3
Mitral-systolic murmur		1
Left presystolic murmur		1
Dull first sound		5
Sharp second sound		2
Not stated		33
		—55
MODE OF DEATH :		
Asthenia		23
Angina pectoris		3
Asphyxia		1
Coma		3
Rupture of heart		14
Rupture of aorta		6
Not stated		5
		—55
STATE OF AORTA :		
Atheromatous		24
Not stated		28
Healthy		3
		—55
STATE OF VALVES :		
Normal		16
Aortic affected		9
Mitral affected		6
Not stated		24
		—55
STATE OF CAVITIES AND WALLS OF HEART :		
Fatty deposit		19
Cavities thinned		18
Cavities thickened		13
Not stated		12
		—55
STATE OF FIBRES :		
Advanced fatty degeneration		50
Granular ditto		5
		—55
STATE OF OTHER ORGANS :		
Fatty liver		7
Fatty kidneys		5
Granular kidneys		2
Coronary arteries diseased		21
Cirrhosis of liver		1
Not stated		19
		—55

TABLE VI.—FATTY HEART.
Dublin Pathological Society (from and including 1854-5).

Author.	Sex & Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Bennett.	F. 24	..	4 years	Dyspnoea; oedema	Occasional systolic murmur at right apex	..	Normal, but congested	Normal	Normal	Fatty degeneration in patches	
Dr. H. Benson	M. 64	Gentleman	..	Dyspnoea on ascending steps; pains in arms and head; fainting	Weak action of heart	Syncope from rupture of left v. on posterior surface	Incipient atheroma	Normal	..	Advanced fatty degeneration	
Dr. Gordon	M. 41	Weak and intermittent pulse; cold face; congested lungs	Bronchial rales	Angina pectoris	Thinned, and nearly converted into fat; left, loaded with fat	..	Congestion of bronchial membrane, & pulmonary apoplexy.
"	M. 14	Immature orphanage	..	Weakness, rowelness; quick, weak pulse; suddenly painful, and loss of consciousness in head motion in right lower limb, followed by gangrene of the toes; sore throat; dyspnoea; pain at precordium	Weak, but regular action of heart; semula feeble, but normal; no murmur	Asphyxia slowly produced	Atheromatous	Normal	..	Fatty degeneration, (granular)	Large emphysematous and fatty liver and kidneys; fatty thrombosis of main artery of right lower limb, from common iliac downwards, with indication of its filling membrane.
Dr. Hayden.	M. 50	Blacksmith	11 months	Irregular, quick and falling pulse; lividity and emaciation	Feeble action and sounds of heart	Asthma	..	Normal	Dilated, hypertrophied and loaded with fat	Fatty degeneration	Kidneys fatty.
"	M. 60	Gunsmith	8 months	General dropsy, including genitals; irregular and intermittent pulse; sighing, and occasional irregularity of breathing; enlargement of liver, and jaundice; visible pulse; hæmoptysis, erysipelas and gangrene of right side of neck	Increased area of precordial dulness; systolic murmur at apex, left axilla and back; diastolic murmur at base, and want of correspondence between radial pulse and action of heart	Asthma	Dilated and atheromatous	Normal; aortic incompetent by dilatation of aorta	Right v. dilated; left v. dilated and thick; heart 2½ oz.	Fatty degeneration, advanced	Liver fatty; right lung hypertrophied; aortic aneurism; right bronchial and bronchial arteries inflamed.
"	M. 69	Cabinet-maker	..	Lividity, oedema, hæmoptysis; falling pulse, enlarged and rough liver; jugular and carotid pulsation	Rapid and irregular action of heart; musical-systolic murmur at apex, and in left axilla and back; loud rales	Coma	..	Normal, except a few spicula at base of aortic	Right v. and a. dilated, and loaded with fat; left v. thickened; heart 18 oz.	Fatty degeneration	Liver enlarged, and cirrhotic; kidneys large and fatty.

TABLE VI.—Continued.

Author.	Sex	Age	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden ..	M.	62	Architect	..	General neuralgic pains, and localised in right mammary region; dry cough	Dull first, and sharp second sound	Rupture of aneurism into pericardium	Atheromatous and dilated in to an aneurism	Normal	Left v. hypertrophied and dilated, and loaded with fat, as were also right a. and v. to greater depth; a thin but healthy stratum of muscular fibre alone remaining on internal surface	Healthy	Aneurism at root of aorta.
Dr. Hewitt ..	F.	Dyspnoea only	None detected, as no cardiac disease was suspected	Rupture of left v. at base posteriorly	..	Normal	..	Fatty degeneration	..
Dr. Jennings	M.	40	Debility, fluttering and irregular pulse, dyspnoea	Weak and irregular action of heart	Asthenia	Atheromatous	Normal	Atrophied, thinned, and covered with fat	Fatty degeneration	Double pulmonary apoplexy.
Dr. H. Kennedy	M.	68	Bronchitis, oedema of feet, regular pulse	Impulse and sounds feeble	Suffocation	..	Normal	Thinned and enlarged, and loaded with fat, especially right v.; adherent and ossified pericardium	Fatty degeneration	Bronchial congestion.
"	M.	65	..	Some months.	Feeble pulse, lividity and anasarca; dyspnoea	Extended precordial dullness	Suffocation	..	Normal	Thinned and loaded with fat; acute pericarditis, with effusion	Fatty degeneration	..
Dr. Kidd ..	F.	23	Dyspnoea, dilated pupils, sinking	..	Asthenia	Thinned and almost entirely replaced by fat over right v.; left v. pale	Fatty degeneration	Liver fatty.
Dr. Law ..	F.	67	Anasarca; dyspnoea	Was in <i>extrema</i> , and no physical examination made	Asphyxia	Excentric hypertrophy of left v.; thinning and dilatation of right v., walls of which were almost entirely replaced by fat	..	Lungs emphysematous.

TABLE VI.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. T. E. Little	M.	65	Gentleman	..	Corpulence; two attacks of oppression and vomiting a few hours before death	..	Syncope	..	Normal, except a few spots of atheroma, and some in coronary arteries	Heart enlarged and loaded with fat; both a. and left v. ruptured on anterior surface near apex, and partially ruptured near the same point	Fatty degeneration, and interstitial deposit	
Dr. McDowell	F.	16	Fever, convalescence, suffocation, catarrh, and sinking	No note	Sinking and asphyxia	Heart small; right v. almost entirely converted into fat; left v. pale	Fatty degeneration	Bronchi congested; air and oil in blood.
Dr. Murney..	F.	60	Bornet-maker	40 years	Recurrent palpitation; debility; arcs; rapid pulse in the paroxysms; 70 ordinarily	Weak sounds; only the first audible in paroxysms; weak impulse; jugular pulsation	Normal	Thinned, and in advanced state of fatty degeneration	Fatty degeneration	General emphysema.
Dr. W. Moore	M.	55	Walter	..	Lividity, dyspnoea, anasarca; faintings; irregular and intermittent pulse	Weak, irregular, and intermittent action of heart	Dilated; right v. nearly replaced by fat	Fatty degeneration	

SYNOPSIS OF TABLE VI.

SEX :		Cases.
Males		12
Females		6
		—18
AGES :		
Under twenty years		2
Twenty to thirty years		2
Forty to fifty "		2
Fifty to sixty "		2
Sixty to seventy "		9
		—18
DURATION OF ILLNESS :		
Minimum	eight months.	
Maximum	forty years.	
SYMPTOMS :		
Faintness, intermittent and irregular pulse, etc.,	in 12	
Paroxysmal dyspnoea	" 1	
Angina (pain, palpitation, and dyspnoea)	" 2	
Dropey	" 8	
Arcus senilis	" 1	
		—18
PHYSICAL SIGNS :		
Irregular action of heart		3
Intermittent ditto		1
Aortic-diastolic murmur		1
Mitral-systolic murmur		3
Dull first sound		7
Sharp second sound		1
Not stated		2
		—18
MODE OF DEATH :		
Asthenia		5
Angina pectoris		1
Asphyxia		4
Coma		1
Rupture of heart		2
Rupture of aorta		1
Not stated		4
		—18
STATE OF AORTA :		
Atheromatous		5
Congested		1
Not stated		12
		—18
STATE OF VALVES :		
Normal		12
Aortic affected		1
Not stated		5
		—18
STATE OF CAVITIES AND WALLS OF HEART :		
Fatty deposit,	in 12	
Cavities thinned	" 10	
Cavities thickened	" 4	
		—18
STATE OF FIBRES :		
Advanced fatty degeneration		14
Granular ditto		1
Healthy		1
Not stated		2
		—18
STATE OF OTHER ORGANS :		
Pulmonary apoplexy		1
Fatty liver		3
Fatty kidneys		2
Cirrhosis of liver		1
Not stated		11
		—18

TABLE VII.
A author's Cases.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Modes of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden...	M.	69	Float driver	..	Chronic cough; orthopnea; recurrent vertigo; pulse 108, regular; rigidity and tortuosity of temporal arteries; urine, sp. gr. 1.025, with a trace of albumen; hæmoptysis; jugular pulsation; oedema; erysipelatous inflammation of genitals, and finally somnolence, and contraction of the pupils	Diffused and feeble cardiac impulse; no apex-pulsation to be felt, a murmur replacing first sound at apex and to left; transmitted systolic murmur at base; weak second sound; signs of pulmonary emphysema	Asthenia	Aorta near ventricle was greatly dilated and aneurysmal in patches; the orifice was not dilated, and the valves were competent	..	Heart enlarged, soft, and flabby; it weighed 25 oz.; right v. covered by a layer of fat two lines thick; an irregular white patch on its surface; right a. and v. dilated, and latter likewise thinned; tricuspid opening dilated, and valves incompetent; right chambers contained a yellow thrombus, which passed through tricuspid opening, and bound up antero-left segment of valve so as to render it incompetent; left a. and v. dilated and thickened; all the valves structurally sound, mitral valves apparently competent	Granular degeneration and obsolescence of fibres of both v., and granular change only of superficial fibres	Liver, spleen, and lungs congested.
"	F.	45	..	Some years	Cough, dyspnoea, debility, weak and intermittent pulse; hæmoptysis	Sharp first sound and accompanying soft murmur at apex	Asthenia	..	Mitral tricuspid bicuspid	Heart large and covered with fat; right v. dilated	Granular stage of fatty degeneration	Pulmonary emphysema and lobular interstitial pneumonia.
"	M.	68	Soft-goods dealer	11 weeks	Fatigues, oedema, quick and irregular pulse; hæmoptysis	Feeble impulse; systolic murmur at left apex; ditto at lower sternum and in ascending aorta; faint second sound; rough systolic murmur (pericardial) over root of pulmonary artery	Asthenia	Atheromatous	Mitral and aortic thick, and the latter calcareous	Heart large and covered with fat; left v. concentrically hypertrophied	Fatty degeneration(?)	Lungs (apices of both) enveloped in false membrane, and right lung congested.
"	M.	66	A porter	12 months	Dyspnoea; oedema; quick and irregular pulse, and venous pulsation in the neck; and, a few days before death, left hemiplegia and inability to articulate	Quick and irregular action of heart; apex displaced to left, and first sound abnormally clear	Exhaustion	..	All the valves normal	Heart large and flabby, with slight deposit of fat; right v. dilated and thin	Advanced fatty degeneration	Blood clot in right anterior lobe of cerebrum, and fibrinous plug in right middle cerebral artery; granular degeneration of the kidneys.

TABLE VII.—Continued.

Author.	*Age	*Sex	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden..	M.	40	Reduced gentleman	Some weeks	Derangement of stomach and "anomalous nervous symptoms"	..	Syncope	Dilated and atheromatous, and lining membrane fissured	All sound, except one segment of mitral, which was slightly thickened	Heart large (18½ ounces) and covered with fat; left v. dilated and thickened	Advanced fatty degeneration, especially of some of the fleshy columns of left v., which were mottled yellow	Lungs congested.
"	M.	70	Tailor	6 years	Dyspnoea; general dropsy; arcus senilis; cough and frothy expectoration; irregular breathing; feeble pulse; rigidity of arteries; throbbing of carotids	Feeble cardiac impulse; systolic murmur at lower sternum; sharp second sound	Syncope	Dilated and atheromatous	Aortic thickened at root; one of mitral opaque			
"	M.	62	Carpenter	12 months	Paroxysmal dyspnoea; "ascending and descending" respiration; failure of pulse; oedema and paralysis of left arm	Weak and rapid action of the heart; clear sounds at lower sternum, and systolic murmur	Asthenia	Greatly dilated, and slightly atheromatous	Mitral and aortic valves thickened and inadequate	Right chambers normal; left v. thickened, except apex, where it was thinned	Fatty degeneration of left v.	Serous effusion into right pleura; other organs not examined.
"	M.	60	Labourer	3 months	Dyspnoea; dizziness; "ascending and descending" respiration; no oedema; somnolence	Rapid and irregular action of the heart; double murmur at base, and systolic only in the carotids; diastolic murmur, and a quad-presystolic murmur at the apex	Asthenia	Dilated and calcareous	Aortic thick & incompetent; watery vegetations at root of mitral, which were otherwise healthy and competent	Heart enlarged (19 ozs.); left v. thickened	Fatty degeneration, advanced in right, & slight in left v.	
"	M.	34	Delirium, tremors; rapid pulse; sleeplessness	No perceptible cardiac impulse; sounds obscure; no murmur	Syncope	..	All healthy	Heart enlarged (19½ ozs.); left v. dilated; heart covered with fat	Granular degeneration of internal layers of left ventricle "Glassy" stage of fatty degeneration	Lungs congested.
"	F.	40	Acute articular rheumatism; great depression and weak pulse	..	Syncope	..	All healthy	Normal as to size, etc.		

TABLE VII.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Hayden..	F.	23	Householder	16 years	General dropsy; dyspnoea, lividity; albuminuria; venous turgescence in neck	Extended precordial dullness; strong impulse behind lower sternum; systolic bellows-murmur at apex, and at base; second sound dull in aorta, but sharp in pulmonary artery	Coma	Narrowed, and of a crimson tint internally	Mitral valves agglutinated and degenerated; mitral ring only narrow; aortic slit; aortic regurgitation; slightly enlarged; right coronary artery not competent	Heart enlarged (13½ oz.); right chambers dilated and thickened; left a. and pulmonary v. thickened; left v. thickened; right auricle and pulmonary orifices dilated	Granular degeneration and loss of transverse striation	Much liquid in the serous cavities of chest, and accumulation, congestion and slight emphysema of lungs; kidneys thick and yellow in cortex.
"	M.	53	Gardener	3 weeks	Recurrent syncope; cough; dyspnoea, and irregularity of breathing	Systolic murmur at lower sternum, and absent of second sound here	Syncope	Greatly dilated, rough, and fissured	Aortic slightly thick and opaque, but competent	Heart enlarged (18 oz.) and fatty on surface	Fatty degeneration and partial obscurity of fibrous sheath	Lungs emphysematous and congested, and traces of former right pneumonia; dilatation of trachea.
"	F.	43	"	Some time	Swelling of feet; weak and variable pulse; convulsive seizure	Apex-beat displaced to left; systolic apex and systolic murmur; later audible in the carotids	Asphyxia after convulsions	Healthy	Anterior segment of mitral thickened, and a flake of fibrin on its ventricular surface which floated in to orifice of aorta.	Hypertrophy and dilatation of left v.; deposit of fat on surface of right v. which was dilated and thin, as was also right a.	Granular degeneration	Serous effusion into pericardium and peritoneum.
"	M.	45	House-painter	9 months	Sudden hemiplegia; epistaxis; vomiting; shortness of breath; pallor; albuminuria, & granular tube-casts; erysipelas; uræmic coma; rapid pulse; rigidity of arteries; alternate dyspnoea and apnoea	Displacement of apex to left; apex systolic murmur; second sound sharp at apex	Coma	Dilated and atheromatous	Fibrous deposit upon the mitral valve	Hypertrophy of the left v.; heart weighed 23½ oz.	Granular degeneration of the heart	Cirrhosis of liver, kidneys, and spleen.
"	F.	18	Orphan child of	Some time	No cardiac impulse; faint sounds		Asphyxia	Normal	Healthy	Hypertrophy with dilatation of left v.; dilatation of ..	Granular degeneration of ..	Granular degeneration of the kidneys; enlargement

Sex, age, occupation.	No.	Signature	Age	Accident	Present condition	Diagnosis	Prognosis	Remarks	Diagnosis	Prognosis	Remarks	Diagnosis	Prognosis	Remarks
"	M. 48	Labourer	3 years	Irregular and suspicious breathing; weak and intermittent pulse; albuminuria and polyuria; edema of feet; vomiting, and diarrhoea.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					
"	M. 56	Farmer	17 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					
"	M. 44	Coachman	2 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					
"	M. 44	Discharged soldier	4 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					
"	M. 43	Peolar	6 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					
"	M. 26	Labourer	26 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	Angina pectoris; recurrent feeling of faintness; edema of feet and legs; orthopnea.	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis.	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnea; hæmatemesis.	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium.	Vertigo; faintness; dyspnoea; visible pulsation at wrist; cough and expectoration.					

SYNOPSIS OF TABLE VII.

SEX :*						Cases.
Males	17
Females	5
						— 22
AGE :						
Under twenty years	1
Twenty to thirty years	1
Thirty to forty	1
Forty to fifty	11
Fifty to sixty	2
Sixty to seventy	5
Seventy to eighty	1
						— 22
DURATION OF ILLNESS :						
Minimum	three weeks.
Maximum	sixteen years.
SYMPTOMS :						
Faintness, intermittent or irregular pulse,	in 16
Paroxysmal dyspnoea	" 11
Angina pectoris (pain, palpitation, and dyspnoea)	" 1
Droopy	" 12
Arcus senilis (noted)	" 1
						— 22
PHYSICAL SIGNS :						
Irregular action of heart,	in 2
Aortic-systolic murmur	" 8
Aortic-diastolic murmur	" 2
Double aortic murmur	" 2
Mitral-systolic murmur	" 7
Left presystolic murmur	" 1(?)
Dull first sound	" 4
Sharp second sound	" 4
						— 22
MODE OF DEATH :						
Asthenia	17
Asphyxia	2
Coma	3
						— 22
STATE OF AORTA :						
Atheromatous	11
Congested	2
Healthy	3
Reduced in calibre	1
Not stated	5
						— 22
STATE OF VALVES :						
Normal,	in 9
Aortic affected	" 8
Mitral affected	" 9
						— 22
STATE OF CAVITIES AND WALLS OF HEART :						
Fatty deposit,	in 11
Walls of chambers thinned	" 9
Walls of chambers thickened	" 14
						— 22
STATE OF FIBRES :						
Advanced fatty degeneration	10
Granular stage	11
Healthy	1
						— 22
STATE OF OTHER ORGANS :						
Fatty liver	2
Fatty kidneys	4
Granular degeneration of kidneys	5
Cirrhosis of Liver	3
Healthy.	8
						— 22

* There was no female over forty-eight years of age, and no male under thirty-four years of age.

The *treatment* of fatty accumulation upon the heart, when this condition can be inferred rather than positively diagnosed, differs in no respect from that of general obesity. The measures taken may be moderately active, if the person under treatment be young and adjudged structurally sound. Thus, if the pulse be strong and regular; the heart, as judged by percussion and auscultation, free from hypertrophy, dilatation, and valvular lesion; the aorta healthy, as determined by the quality of the cardiac sounds in the ascending aorta, and the character of the respiration; and the kidneys free from disease, as shown by a careful general and microscopical examination of the urine, under such circumstances, active exercise may be enjoined, especially that which quickens respiration and consumes heat-producing tissue; such as quick walking up hill, the use of the dumb-bells or quoits, racket-playing, etc. At the same time the use of hydrocarbons in food and drink must be restricted, especially sugar, fats, milk, and malt liquors. For the same reason rum and liqueurs ought to be forbidden. Wholesome nitrogenous food should be taken frequently to anticipate a craving appetite, but always short of satiety. A bath, either cold or tepid, should be frequently taken; the bowels should be well moved at least once daily, and the period assigned to sleep should not exceed seven hours out of the twenty-four. Alkaline waters, natural and artificial, such as those of Vichy and Kissingen, Seltzer, etc., may be freely drunk. This plan, by which the adipose tissue is *gradually* reduced, and then kept within moderate limits, is very different from the extreme and heroic system of Banting, by which it was proposed to *rapidly* dissipate fatty accumulations.

The system of Banting involved no little danger to the integrity of structures and organs, especially those subject to periodic and frequent distention, such as the heart and arteries, by the attenuation and weakening of their walls which necessarily follow the rapid removal of interstitial and superficial fat. There is, moreover, considerable danger of actual degeneration of tissue from abrupt reduction of the wonted supply of pabulum. Hence, Bantingism should, under all circumstances, be discontinued, even in the young and healthy. The plan thus briefly sketched should be put in operation in all cases, but modified according to age and

other circumstances, where there is a tendency to adipose development. Thus, in middle age, especially where there is such evidence of cardiac weakness as that afforded by feeble impulse and radial pulse, and palpitation and dyspnoea after a moderate walk, pedestrian exercise should be limited, but regular, and always, if possible, on level ground, in the open air, and short of fatigue. Even in such cases, if there be not conclusive evidence of structural change and advanced attenuation of the heart, moderate and regular exercise on foot, such as walking or playing billiards, should be recommended, as conducing not only to the reduction and limitation of fat, but to the healthy nutrition of the tissues of the body generally. I have already stated as my opinion, and I would repeat it here, that of the rare and peculiar condition of the heart in which its proper structure is represented, but in different proportions in the several cavities, by only a thin layer of muscle internally, the greater portion of the substance of its walls having been replaced by an ingrowth of fat from the outer surface, the thin residuum of muscle being, however, *structurally* sound, there is absolutely no positive evidence, general or physical (see "Aneurism," Mr. B.).

It would seem that in such cases, provided always the duty imposed upon the heart by exercise or excitement be not even *once* in excess of the ordinary routine, that the attenuated muscular wall is enabled by the soundness of its structure, and by the mechanical support which it receives on the outside from a thick and solid layer of adeps to perform its function without faltering. Where this condition is suspected, from the existence of a feeble but regular pulse, and the absence of precordial impulse conjoined with general obesity, positive diagnosis being, in the present state of knowledge, impracticable, physical effort and excitement of every kind should be strictly prohibited. Measures should be taken to improve the general nutrition in the hope of advancing that of the heart; and tonics, such as iron, quinine, and strychnia, should be given with a view to strengthening the portion of muscular substance which remains.

Where fatty degeneration of the heart actually exists, there cannot be a hope of equal benefit from treatment. Tissue is now in process of retrograde metamorphosis, and the utmost that can be hoped from treatment is to stay that fatal process, whilst

guarding against accidents from the changes which it has already wrought. Digestion should be promoted, the appetite, if possible, improved, and the bowels, which are usually confined, should be regulated by means of mild saline aperients. Medicines capable, at the same time, of improving the appetite and promoting the nutrition of the muscles by imparting tone and vigour to their action, should be steadily and perseveringly administered. Of these, the best are quinia and strychnia, given in moderate doses, and combined with iron where the anæmic aspect is presented. I usually direct the following: *R.* Sulph. quiniæ, grs. xxiv; Acid. sulphur. dil., ℥xxx; Liquor. strychniæ (B. P.), ℥xlviij; Sulph. ferri granulat., grs. viii; Syrupi flor. aurant., ʒiv; Aquæ font., ad ʒviij. A tablespoonful to be taken thrice daily.

Should there be evidence of sanguineous engorgement of the liver and of fatty accumulation in its cells, as furnished by enlargement and tenderness of that organ, and a jaundiced tint of the skin and conjunctivæ, then an occasional dose of blue pill, combined or not with resin of podophyllin according to the state of the bowels, should be directed. In the treatment of dropsy dependent upon fatty degeneration of the heart, a combination usually consecutive to chronic disease of the kidneys, reliance must be placed mainly upon active purgation with saline aperients, and the dry air-bath. Where the kidneys are merely congested, or secondarily engaged, the urine exhibiting slight opalescence when heated or tested with nitric acid, the specific gravity being 1.020 or upwards, after dry cupping and warm poultices to the loins, diuretics should be given. Of the latter, decidedly the best is digitalis; because it is not only the most efficacious as such, but also because it is the most active and reliable tonic stimulant of the heart which we possess. It is still more decidedly indicated if, in addition to the presence of dropsical effusion, the action of the heart be faltering and irregular. The effect of this medicine in restoring regularity of action to the heart, and increasing its contractile vigour and force, entitles it to the first place as a cardiac tonic. I prefer the tincture to all other preparations of this drug, and usually prescribe it as follows: *R.* Tinct. digitalis, ℥lxxx; Spirit. æther. nitros., ʒij; Aquæ font., ad ʒviij. An ounce every third hour. It is in cases of the former kind, namely, primary disease of the kidneys, fol-

lowed in succession by hypertrophy and fatty degeneration of the heart with atheromatous change of the arteries, that the most formidable of the complications of fatty disease of the heart, gangrene of the lower extremities, is most frequently met with. There is previously considerable œdema of the feet and legs with tension of the skin; an erysipelatous blush then appears upon the dorsum of one or both feet, or above the ankles, attended with elevation of temperature, and here one or more bullæ filled with amber-coloured serum, are formed; the surrounding surface is now congested, the temperature falls, and gangrene rapidly spreads from the bullæ as from so many centres. I have reason to believe that in such cases there is always the antecedent change of aortic arteritis which is intimately associated with the immediate and determining cause of gangrene; but, inasmuch as acute inflammation of the lining membrane of the aorta is always a sequela of atheromatous conversion of its walls, and as the latter is usually accompanied by dilatation of the vessel, we might expect to find rhythmical dyspnoea, or irregularity of breathing, a prominent feature of those cases in which gangrene of the lower limbs is a final symptom; and so it is. In all the examples of this truly formidable complication which have come under my notice, there were present rhythmical dyspnoea, and the sharp, clicking, second sound in the ascending aorta, indicative of atheromatous conversion of its walls. The gangrene is, I believe, immediately dependent upon local arteritis and thrombosis. If tension of the integument be excessive, acupuncture may be practised, or the skin, where most on the stretch, may be punctured freely with the point of a lancet at distant intervals; the seat of most active inflammation being, however, avoided. A lotion, consisting of 3ij of Goulard's solution to 3x of water, should be kept constantly applied to the inflamed portion of the skin, whilst those parts which are already livid, and in which the temperature has fallen below the normal standard, should be wrapped in cotton wadding, or soft wool.

In the treatment of the distressing form of dyspnoea, designated "of ascending and descending rhythm," I have found the inhalation of nitrite of amyl the most efficacious means of relief. Ten or twelve drops may be received into the folds of a pocket-handkerchief or napkin, and freely inhaled by the patient; or a

few whiffs may be taken, at the acme of respiratory distress, directly from a bottle containing the nitrite. The effect has been to mitigate the feeling of want of air, and to slow the respiration. The peculiar effect of this agent is to relax the systemic capillaries and increase the volume of blood circulating in the tissues. It was a knowledge of this action of the nitrite of amyl, that first suggested to me a trial of it for the relief of the distressing symptom under notice, which depends, as already shown, upon failure of tissue-respiration.

The treatment of the closely allied symptom of angina pectoris, so often presented in connexion with hypertrophy and fatty disease of the heart with atheroma of the arteries, especially in gouty subjects, should be conducted on the same principle; namely, that of promoting capillary circulation, and relieving the distended heart. For the accomplishment of this twofold purpose, I am not acquainted with any measures more efficacious than dry cupping over the chest, warm stimulating applications to the feet and legs, such as hot mustard baths or sinapisms, and nitrite of amyl given by inhalation, as described in the preceding paragraph. Dr. Strange, of Worcester, has found the hydrate of chloral, in doses of grs. xxv-xxx, most efficacious in the treatment of angina pectoris. It should be given in the above mentioned dose every hour, till relief, which usually follows the first dose, is obtained. Two cases are mentioned by Dr. Strange in support of his statement, and in one of these, a dose of chloral was given nightly for four months, with a few omissions; on these latter occasions only the angina returned.* I cannot speak from personal experience of the treatment of angina with hydrate of chloral, but I would not hesitate to make trial of it, as recommended by Dr. Strange. The intercurrent angina of fatty heart is frequently dependent upon sub-acute aortitis; such cases are characterized by a feeling of great constriction at the upper portion of the sternum, and are best treated by leeching in this situation, and subsequently by the administration of small and repeated doses of grey, and antimonial powder.

A six weeks' residence at Kissingen or Harrogate, and a course of mild alkaline chalybeate waters, would be eminently condu-

* *Medical Times and Gazette*, September 24th, 1870.

cive to the restoration of tone and vigour to those fibres of the heart which are suffering from feeble nutrition, but not yet actually in process of fatty change. Such fibres, which are characterized by granular dotting at intervals, transverse striation being still manifest over the greater portion of their length, may be found in greater or less number even in hearts most advanced in fatty metamorphosis. They are the representatives of healthy structure in the organ, and the sole agents in carrying on the circulation. Hence the importance of arresting the further process of change in them by promoting their nutrition.

I now proceed to submit examples of the condition discussed at such great length in the preceding pages.

CASE XLII.—*Pulmonary Emphysema and Interstitial Pneumonia; Mitral Regurgitation; Death. Fatty Accumulation upon the Heart, and Granular Degeneration of its Substance.*

Susanna H., aged forty-five years, was admitted into the Mater Misericordiæ Hospital, January 20th, 1865, suffering from cough with great dyspnoea and debility. Pulse 114, very weak, and intermittent. The cervical veins were distended, and there was venous and arterial pulsation in the neck. At the apex of the heart the first sound was clear and accompanied by a soft systolic murmur. Alcoholic stimulants were freely administered, and under their use her general condition was improved.

On the 14th of February, whilst the breathing was less embarrassed, the pulse was scarcely to be felt, and on the 15th streaks of florid blood appeared in the sputa; an event which, as she then admitted, had frequently occurred before. The apex-murmur, which up to this date continued to be audible, was not to be heard. During the two days immediately preceding her death, which took place on the 20th, no pulse could be felt at the wrist.

The body was examined on the following day. Both lungs were partially emphysematous, and several patches of interstitial pneumonia, in its early stage, were found in them. The bronchial membrane was congested, and the bronchial tubes contained much viscid mucus. There were several ounces of serum in the sac of the pericardium, and the heart, especially at its base, was

covered with a thick layer of fat. The right chambers were dilated and filled with dark coagulated blood. The left ventricle was somewhat thickened, but not dilated, and the mitral valve was tuberculated upon the edge. The aortic orifice was slightly contracted, but the valves were healthy and competent. No striæ were visible in the muscular fibres of the heart examined microscopically. The fibres were pale, and exhibited lines of refractile granules. To this case is appended in my register the following note, which I copy *verbatim*: "This with some other similar cases, viz., of partial emphysema with congestive bronchitis, suggests the reflection, that in such the aggravated dyspnoea, failure of circulation, and anasarca, are due, not so much to the affection of the lungs, as to fatty and weak heart, which I have always found to be an associated condition. Hence the benefit derived from the free use of brandy and other stimulants." In such cases, likewise, mitral reflux, not dependent upon valvular disease, is of frequent occurrence, from weakness and yielding of the walls of the left ventricle at the acme of systole.

CASE XLIII.—*Aneurism of the Ascending Portion of the Arch of the Aorta; Inadequacy of the Aortic Valves; Hypertrophy and Fatty Degeneration of the Heart; Death.*

Rev. Mr. R., aged forty-five years, was received into the Mater Misericordiæ Hospital as a private patient on the 2nd March, 1865. He was a large man, and had been very corpulent. Had irregular and intermitting pulse for the last ten years, and was told that he had fatty disease of the heart.

In July, 1864, he had one or two attacks of vertigo, and in November of the same year he caught cold, and had a bad attack of bronchitis; his breathing then became short for the first time.

On the night of January 15th, 1865, when about retiring to bed, he felt a sensation of tightness about the upper part of the sternum, accompanied with hurried and difficult respiration, and with great excitement and alarm; he did not venture to go to bed that night, and he has not been since able to lie down. The feet began to swell about the 15th February.

When admitted, he was remarkably pale, and rather thin; the pupils were much contracted, even in the shade; the figure was bent, and there was complete orthopnoea. He experienced most relief by leaning forward in the sitting posture. The lower limbs were throughout cedematous, and pitted on pressure, but were free from congestion. Urine loaded with lithate of ammonia, sp. gr. 1.027, and passed in small quantity; only twenty-two ounces in the twenty-four hours; it contained a trace of albumen. Bowels rather confined; pulse 90 to 96, full, soft, irregular, and occasionally intermitting, but not "late" in relation to the ventricular contraction. Cardiac impulse feeble; first sound ill pronounced. A sharp, whizzing murmur replaced the second sound at midsternum, was traceable upwards and to the right with increasing loudness, and attained its greatest intensity at the upper part of the sternum; in this situation there was a considerable circumscribed prominence engaging the right side of the sternum, and three superior costo-sternal articulations of the same side. Over this prominence, which was comparatively dull on percussion, the diastolic murmur, as already stated, was loudest; it extended into the right subclavicular region, but not into the left. Two sounds were heard over this prominence; viz., one corresponding to the first sound of the heart and similar to it in character, the second being the diastolic murmur, heard in its greatest intensity. The most careful examination failed to detect impulse over the prominence, or elsewhere, save in the normal situation of the heart. Neither impulse nor murmur was perceptible in the back. Respiration 30, feeble on right side, loud on left, and accompanied with coarse râles over both bases posteriorly. No visible pulsation of the arteries, and no thoracic pain, but a feeling of "tightness" behind the fourchette of the sternum. No distention of the superficial veins of the chest, but the right external jugular was distended at its junction with the subclavian. No difference between the radial pulse on the opposite sides. Diagnosis: Aneurism of the upper portion of the ascending aorta pressing on the right bronchus; this aneurism is probably false, one or more of the coats of the vessel having given way on the night of January 15th; aortic valves inadequate, most probably by simple dilatation of the orifice; and finally, hypertrophy of the heart from antecedent

atheroma of the aorta; fatty degeneration of the heart, in a not advanced stage; and engorgement of the liver.

On the 9th March a feeble impulse was felt over the sternal prominence at the end of expiration, the patient having on the preceding night experienced several attacks of paroxysmal dyspnoea. These attacks were readily brought on by mental excitement or physical exertion of any kind, and were promptly relieved by the application of a few leeches. He was usually incoherent at night, but collected during the day. Œdema extending upwards. Under the use of tincture of digitalis in doses of ℥x, with an equal quantity of spirit of nitrous ether, and of elaterium and blue pill in small doses, the urine was increased in quantity, the œdema was somewhat reduced, and the pulse-rate brought down to 66-72.

On the 22nd March the pulse was very irregular; there were 14 beats within the first recorded period of ten seconds, 17 in the second, and 15 in the third corresponding period. He had a paroxysm of dyspnoea, during which it was thought he would have died; it came on without any assignable cause, was accompanied by dry, hacking cough, and lasted two hours.

On the 23rd, œdema had extended to the abdomen, which contained some liquid, and fine crepitation was audible over the base of both lungs. There was likewise persistent dry cough. Infusion of digitalis was now given, in half ounce doses, with spirit of juniper and chloric ether; this produced copious diuresis, and was followed by some relief from the dyspnoea. The legs were subsequently punctured to avert gangrene of the over-stretched skin; on the 3rd April he spat some blood.

April 5th. Pulse 108, full and bounding. The urine contained a larger proportion of albumen, and the right foot, and lower portion of right leg, were tender and erythematous; œdema much reduced; heart's action strong; both sounds audible, but without murmur; two sounds were likewise audible over the sternal prominence, *but no murmur*; no dysphagia.

On the 6th, the murmur was again audible at the base of the heart, and over the sternal prominence, as before.

On the 14th, an eruption of the character of discrete variola appeared on the lower half of the right arm, and about the

elbow, but was strictly confined to these situations; the contents became opaque, and the pustules began to fade on the following day; and then, for the first time, there was visible pulsation in the radial arteries and carotids.

26th. The scrotum, which was greatly distended with serum, was punctured to-day. Respiration 36, performed rapidly, and at long intervals; great cardiac distress, and murmur nowhere audible; respiratory murmur very feeble over right front; loud over left.

He died on the 3rd May, and permission to examine the body could not be obtained. I think, however, the diagnosis made was fully warranted by the symptoms and physical signs exhibited. As the case is so full in detail, and in many respects so typical of its kind, I have deemed it worthy of publication, even without the proof afforded by dissection.

In connexion with the preceding case, the next, which in many respects resembles it, although differing in some particulars, may be given.

CASE XLIV.—*Fatty Degeneration of the Heart without Valvular Lesion.*

The Rev. Mr. P., aged thirty-five years, visited me on the 14th February, 1873. He was a curate, of very active habits and great zeal, and located in a mining district in the south of Ireland, where the vice of drunkenness prevailed to an alarming extent. This he undertook to combat with all his power. With this object in view, he established a temperance society, and hired a room, in which he lectured to the members on the evils of intemperance for several hours, three or four times in the week; pursued them into their haunts, and, being a strong and resolute man, frequently used physical force to drag them out of the public houses. On one occasion whilst so engaged he got very roughly handled. He suffered greatly in mind at the misfortunes of those whom he laboured to reform, and frequently felt quite exhausted after his exertions. About two years prior to his visit to me, he began to experience oppression of the chest with palpitation, and some difficulty of breathing, but without cough.

During the succeeding twelve months he had several attacks of syncope; and three weeks before I saw him, he felt faint from the effects of a hard morning's work, and a rapid walk of some miles after breakfast. The heart, on this occasion, beat with great violence, and he was bewildered and wandered about the house without knowing why or where he went. This state lapsed into complete syncope in which it was thought he would have died, and during which, according to the report of his attendants, he was deadly pale. He declared, however, that neither in this attack, nor in any of those which preceded it, did he entirely lose consciousness.

At the date of his visit to me, the slightest exertion or emotion sufficed to bring on fluttering of the heart, oppression, and vertigo; he had lost flesh, but was still corpulent, though flabby. Pulse 84, and regular but weak; respiration regular and tranquil; skin of an olive tint; liver somewhat enlarged and tender; bowels constipated; urine depositing lithates at intervals; no œdema; no cardiac impulse to be felt; first sound faint and dull, and heard best in normal situation of apex; second sound morbidly clear at base, and in line of ascending aorta. No murmur anywhere to be heard, and no detectable rigidity or tortuosity of the superficial arteries.

The diagnosis made was as above stated, and the treatment consisted in ℥ij of liquor of strychnine, with grs. iiss of sulphate of quinine, thrice daily; a pill containing grs. ij of extract of aloes, gr. j of extract of hyoscyamus, and gr. ss of extract of nux vomica every night, or every alternate night, as required. Moderate exercise on foot, short of fatigue, was enjoined, half a wineglassful of whiskey with water to be taken twice or thrice daily; if threatened with syncope, a little brandy and water, together with a draught consisting of sulphuric ether ℥lx, and laudanum ℥xx, which he is to have ready at hand. Mental excitement, and the use of fatty food, was to be strictly avoided.

Two months later, Dr. O. Hanlon of Castlecomer, the Rev. Mr. P.'s ordinary medical attendant, reported to me that since his return to the country, in February, this gentleman continued to perform the duties of a very arduous mission, and had had no return of his previous symptoms.

In August, after a further interval of three months, I learned that he had been recently transferred to a new sphere of missionary labour.

CASE XLV.—*Fainting Fits; Œdema; Quick and Irregular Pulse; Hæmoptysis; Feeble Impulse and Systolic Murmur at Left Apex, at Lower Sternum, and in Ascending Aorta; Rough Systolic Murmur at Root of Pulmonary Artery; Death. Atheroma of Aorta; Mitral and Aortic Valves Thickened, and latter Calcareous; Heart Large and Covered with Fat, and Fatty Degeneration of its Substance (?) Right Lung Congested, and both Lungs partially Enveloped in False Membrane.*

Owen K., a travelling dealer in soft goods, aged sixty-eight years, admitted into Hospital, March 11th, 1865. Health reported good up to eleven weeks before, when he had an attack of bronchitis. Five weeks later, whilst getting out of bed to pass water, he felt a swimming in his head, and would have fallen had he not been supported. He remained in a state of half consciousness for forty-eight hours after this attack, and had another of a similar kind, but milder and shorter, consisting only of vertigo, on the Sunday preceding his admittance. Œdema of the lower limbs appeared seven weeks ago. He is pale; arcus senilis well marked; feet and legs much swollen, and feet thickly mottled with purpuric spots. Pulse 126, moderately full, and slightly irregular; respiration 24; cardiac impulse feeble. A loud bellows-murmur replaced the first sound at the left apex. A murmur of similar rhythm was likewise audible over the lower part of the sternum, and was traceable upwards in the course of the aorta to the second right chondro-sternal articulation, but less distinctly. The second sound was free from murmur, but ill pronounced, except above and to the right of the sternum, where it was distinctly heard. Respiration was feeble all over the chest, and associated with coarse crepitant râles over the bases of both lungs posteriorly. Cardiac murmur not audible behind. He declared that his breathing had never been irregular, nor was there, during the fainting fits, any cardiac distress or palpitation.

Y. A. S. S. I.

The diagnosis made at this date was regurgitation at the mitral, and obstruction at the aortic orifice, hypertrophy of the left ventricle, and a fatty state of the heart. To have nutritious diet and four ounces of wine daily, and a mild aperient as required.

13th. Pulse 102, slightly irregular, as is likewise the action of the heart. The second sound was accentuated behind the right second costal cartilage, where the systolic murmur was remarkably rough; slight *frémissement* over the heart during expiration; urine, sp. gr. 1·012, containing albumen in small quantity, and amounting to forty-eight ounces in twenty-four hours. To have tincture of the perchloride of iron and chloric ether, of each ʒiij, with infusion of quassia to ʒvii; a tablespoonful to be taken thrice daily. After a few days this medicine disagreed with the stomach, and compound iron mixture was substituted for it.

Still later (28th), citrate of iron and quinine was given in doses of grs. ij.

On the 30th, the pulse was 96, and regular; vertigo on assuming the erect posture. Over left apex a systolic murmur was heard, but no sound; over the base of the ensiform cartilage a systolic murmur and both sounds of the heart were audible.

On the 4th April he had an epileptiform fit, lasting half an hour, during which he lost consciousness and bit his lip; the pupils were dilated; the pulse was full, but not quick, and the cervical veins were engorged, but there was neither muscular spasm nor foaming at the mouth. Previous to the attack he was in a state of extreme nervousness, which he regarded as premonitory of it.

8th. Urine passed in large quantity, containing much albumen, and neutral in reaction, sp. gr. 1·012. Murmur transmitted into right carotid.

11th. Much improved; pulse 84, and strong; appetite and sleep much better; bowels regular, swelling of feet much diminished, and purpuric mottling has nearly disappeared.

13th. Had three attacks of hæmoptysis; viz., at 9, P.M., and at 1 and 6, A.M. In the second attack he threw up more than a pint of florid blood, and was nearly suffocated. Pulse 114, full; respiration 36; base of both lungs dull behind, with coarse crepitation audible, especially on the left side; feet not swollen,

but purpuric mottling now present in an extreme degree. He apprehends another attack in the course of to-night, which he says "will carry him off." To have grs. v of acetate of lead, and gr. iss of acetate of morphia, in water and distilled vinegar, every fourth hour. To be dry cupped over base of both lungs posteriorly, and to have cold drinks.

16th. Had a slight attack of hæmoptysis, accompanied with dyspnœa and excitement at a-quarter past 7, A.M.

25th. Very weak and unable to leave bed; no swelling or discoloration of feet; pulse 108, weak, but regular; respiration 36, strong and visible pulsation of carotids; a harsh murmur with the first sound, and very superficial, heard over the left third costal cartilage at its junction with the sternum, suspected to originate in, or upon the root of, the pulmonary artery. Other cardiac phenomena as before.

28th. Is incoherent and sinking. Over the sternal end of the left third costal cartilage, but loudest half an inch below and to the right of this point, a loud grating murmur is heard with the first sound.

He died on the 29th April, at 1, P.M.

The lungs, heart, and great vessels were removed a few hours after death, and examined on the 2nd May. There was much fat in the anterior mediastinum and around the pericardium; there was likewise a large deposit of fat on the exterior of the heart. Three "white spots" of considerable size were discovered; one about the size of a crown-piece on the anterior surface of the right ventricle, another somewhat smaller on its inferior surface, and a third as large as a florin-piece on the anterior surface of the pulmonary artery, at its origin from the right ventricle. This, no doubt, was the cause of the rough grating murmur heard on the 25th and 28th April, over the sternal extremity of the left third costal cartilage; right cavities not altered; pulmonary artery and its valves healthy; left ventricle greatly hypertrophied, and its cavity diminished; mitral valves much thickened, but no deposit upon them. Aortic valves competent to retain water poured into the aorta, but rough with calcareous deposit. Lining membrane of aorta bright red, and interspersed with yellow patches of atheroma. It was rough, and in a more advanced state of

atheromatous change, at the origin of the great vessels, and in the lower wall of the transverse portion of the arch. The apices of both lungs were encased in a thick layer of false membrane; that on the right side was several lines thick, and the parenchyma of the lung beneath was solidified, apparently by contraction of the enveloping false membrane. The lower portion of the right lung was congested, and likewise covered by a layer of false membrane. Other organs not examined. Owing to an oversight, the muscular substance of the heart was not examined microscopically.

CASE XLVI.—*Dyspnœa ; Œdema ; Quick and Irregular Action of the Heart ; Venous Pulsation in the Neck ; and, eight days before Death, Left Hemiplegia and Partial Aphasia ; Displacement of the Apex of the Heart to the Left, and the First Sound Abnormally Clear ; no Murmur. Extravasation of Blood into the Right Anterior Lobe of the Cerebrum, and Plugging of the Right Middle Cerebral Artery ; Granular Degeneration of the Kidneys ; Enlargement and Fatty Degeneration of the Heart ; Dilatation and Thinning of the Right Ventricle ; Valves all Sound.*

Michael T., aged sixty-six years, a porter, of temperate habits, was admitted into hospital, September 25th, 1865. About two years previously, having been much exposed to wet and cold, he complained of cough. Twelve months later, his breathing became very much oppressed, and his feet began to swell.

On examination, his condition was found to be as follows: There was œdema of the face and lower extremities; the action of the heart was quick (120 in the minute), and irregular; the apex was displaced to the left, and the first sound was abnormally clear. There was dulness on percussion, and inspiratory wheezing, over both lungs posteriorly. Marked pulsation of the superficial cervical veins, synchronous with ventricular systole, was visible during inspiration. Urine diminished in quantity, acid in reaction, sp. gr. 1·010, and without a trace of albumen. To have thrice daily a tablespoonful of a mixture consisting of Iodide of potassium, ℥iss; Acetate of potash, ℥ij; Infusion of gentian, to ℥viij.

October 3rd. Breathing less oppressed; urine increased in quantity (a quart last night) and neutral in reaction. On the following night the urine passed amounted to two quarts, and the night of the 5th the quantity was still greater. There was less tumefaction of the face, and the cedema of the legs had quite disappeared.

22nd. Whilst returning from the water-closet he became suddenly hemiplegic on the left side of the body; the left side of the face was but partially affected, the patient being able to close the eye and corrugate the forehead on that side. Consciousness was perfect, as was likewise the power of deglutition, but there was almost total inability to articulate. No paralysis of the bladder or rectum.

The patient died on the 30th October, and the body was removed for dissection to the Medical School, Cecilia-street, where a careful examination of it was made. The right hemisphere of the cerebrum was soft, and in excess of the left hemisphere in volume. In the upper part of the right anterior lobe there was a large blood-clot, from which blood had been diffused into the surrounding brain substance, breaking up the latter within a radius of an inch and a-half. The corpus striatum and other parts were unaffected. The right middle cerebral artery was so plugged with fibrin, that the ordinary coloured injection used in the dissecting room could not pass, under high pressure. The kidneys were smaller than natural, and the cortex of the left was so attenuated that the Malpighian pyramids had all but come to the surface. The heart was large, soft, and so flabby, that when placed upon the table, it expanded by its own weight, and became quite flat; it was likewise torn with great facility. There was only a very slight deposit of superficial fat. The right ventricle was dilated, and its walls were remarkably thin. The left cavities and all the valves were normal. Under the microscope, the fibres of the left ventricle exhibited lines of oil dots in place of the normal fibrils. In one situation only the transverse striæ were visible.

CASE XLVII.—*Recurrent Angina and Hæmoptysis; Irregular and Intermittent Pulse; Edema; Albuminuria and Slight Jaundice; Feeble Cardiac Impulse and Sounds; Irregularity of Breathing; Gangrene of the Foot and Leg on one Side; Coma; Death. Diagnosis: Dilatation and Atheromatous Change of the Aorta; Enlargement and Fatty Degeneration of the Heart, and Accumulation of Fat upon its Surface; Atheromatous or Calcareous Change of the Smaller Arteries, and Thrombosis of those of the Right Foot and Leg.*

J. N. W., a medical man in extensive practice, aged sixty-one years, large and corpulent, consulted me October 22nd, 1866. Five years previously, whilst ascending a steep incline, he complained of shortness of breath, and spat a little blood. Two years later he coughed up a considerable quantity of blood, and was bled from the arm to eight ounces. Three weeks previous to my visit, after rapidly ascending a flight of stairs to attend an urgent call, he suffered extreme dyspnœa, was obliged to sit down and have the window thrown open in order to catch his breath, and spat some blood.

He had occasionally suffered from derangement of liver and kidneys, the urine being loaded with lithates; and once, very recently, from acute pain in the right lumbar region, which lasted but a short time, and terminated with a copious flow of urine loaded with lithates.

For some days prior to my visit he had been suffering from paroxysms of dyspnœa, accompanied by a feeling of great oppression in the region of the heart. The face and conjunctivæ were slightly jaundiced; liver much enlarged and tender; and pressure at the epigastrium aggravated the feeling of oppression in the region of the heart. The stomach and bowels were distended with flatus; the bowels were confined, and were moved only by means of aperients. The stools were ochrey in colour; urine, passed in small quantity, was clear and amber-coloured at my first visit, but on the following day, fawn-coloured and muddy; in sp. gr. it was 1·020, acid, containing lithates, and a small proportion of albumen. Pulse 114, small, and rather

weak, but regular; according to his own report it has been occasionally intermittent. Heart's action obscure; no impulse perceptible, and precordial dulness extended generally. The first sound was ill pronounced and masked, but free from murmur; second sound in some degree disintegrated and muffled; no impulse anywhere discoverable over the chest. The radial pulses and the pupils were respectively equal, and no dysphagia existed. In the course of the preceding few nights he had got some hours of broken sleep in an easy chair, with the help of 3ss of liquor of the hydrochlorate of morphia. To take daily six pills, containing each gr. iss of squill and of mercury with chalk, and grs. ij of dried soda. A dozen leeches to be applied, in two relays, over the liver, and then a warm poultice. Mercurial ointment was subsequently applied freely in the region of the liver. The use of liquids was restricted. Beef tea and bread crumb were ordered as food, and a small quantity of whiskey and water was given occasionally, when debility seemed to demand it.

25th. 4 P.M., pulse feeble, and occasionally intermitting. In the course of this evening, whilst in bed and at rest, he had a slight attack of vertigo, accompanied with momentary loss of consciousness. Sir D. Corrigan saw him, and coincided in my view of the case.

On the 27th there was slight mercurial fetor; he had several hours of undisturbed sleep the preceding night, after a large blister had been applied over the sternum. To have grs. ij of calomel every sixth hour.

29th. Considerable œdema of lower limbs; had a severe paroxysm of dyspnoea in the course of the preceding night.

November 1st. Since last report he has been improving steadily. He slept well the preceding night in the horizontal posture; abdomen much reduced in girth, and hepatic tenderness all but removed. Pulse regular, but weak; tongue clean, and passed in the preceding twenty-four hours; it was clearer; appetite improved, and patient more comfortable.

It had been ascertained that he was to leave the hospital in a middle stage of the disease.

ill, having been seized with a paroxysm of dyspnœa of unusual severity.

On the 3rd, the pulse was weak and intermittent, and the cedema of the lower extremities was increased.

On the 4th, he became livid, pulseless, and slightly incoherent, and was again visited by Sir D. Corrigan in consultation with myself and his son, a medical officer in the army.

On the 5th, large bullæ had formed on the feet and legs; there were repeated paroxysms of dyspnœa, in which the breathing became gradually more and more rapid, and then as gradually subsided to the normal standard; pupils greatly contracted.

7th. Pulse irregular, intermittent, and weak; pupils contracted; heart-sounds obscure and feeble, but without murmur. He is now unable to get into bed, obtaining only a few snatches of sleep in an arm chair. Feet, legs, and thighs highly cedematous, depressed in temperature, and, as high as the knees, studded with large bullæ containing clear serum. Cannot take food of any kind, except a little egg-flip; is much troubled with hiccough and bilious vomiting.

During the 6th and 7th, there was copious diuresis without deposit, the quantity passed in twenty-four hours amounting to eighty ounces.

Diuresis continued on the 8th; the bullæ had given way, and there was a free discharge of serum from them. The pulse was irregular, intermittent, and barely to be felt, yet the patient declared he felt better.

On the 9th, he was comatose and pulseless; the thighs were greatly swollen, and on the inner aspect of the right, there was a large patch of inflamed surface, of a yellow-red tint.

11th, the right foot and leg, half way to the knee, were gangrene; but no further change had taken place. He was conscious, and had within the preceding twenty-four hours repeated attacks of dyspnœa, attended with livor and cyanosis of the eye-balls. I witnessed one of these attacks of ten minutes. They came whilst the patient lay in bed,

and died, and no examination of

the body was made. The diagnosis given in the title of this case, though unsupported by the evidence of *post mortem* examination, is, I think, fully warranted by the symptoms and signs exhibited, as well as by its analogy to other cases published in this list, in which the diagnosis was confirmed by the crucial test of dissection.

CASE XLVIII.—*Derangement of Stomach, and Anomalous Nervous Symptoms; Sudden Death. Congestion of Lungs; Enlargement and Fatty Growth upon, and Advanced Fatty Degeneration of, the Heart; Excentric Hypertrophy of the Left Ventricle; Dilatation and Advanced Atheromatous Change of the Aorta.*

Robert C., aged forty years, a respectable man, but reduced in circumstances, who had been for some weeks attending at the Mater Misericordiæ Hospital as an extern patient for derangement of bowels and anomalous nervous symptoms, presented himself at the usual hour on Wednesday, July 14th, 1869, in his ordinary state of health, and making no complaint of any particular ailment. On the evening of that day he was carried to the hospital by a person who had found him in an adjoining field, in a state of insensibility. When received into hospital he was quite unconscious, pallid, cold, and pulseless; he died shortly after his arrival.

The lungs were congested, but resonant. The heart large, and covered, on right side especially, with a thick layer of fat; it weighed eighteen and a-half ounces, and expanded by its own weight when laid upon the table. The right ventricle was dilated, but not thinned, and the superficial fat had penetrated nearly to the internal surface at the apex. The left ventricle was dilated and hypertrophied; several of the fleshy columns were dotted over with large yellow spots, and a section of one of them, which presented this appearance over about one-eighth of an inch of its length, exhibited its entire substance, all but a mere shell, converted into soft fat. Microscopically examined, the walls of the heart were found in an advanced stage of fatty degeneration.* The valves were all healthy and competent, with

* By an oversight, the particulars of the microscopic examination were not noted.

the exception of the anterior-right curtain of the mitral, which was a little thickened on its free margin. The arch of the aorta, especially the ascending portion, was much dilated, rugose on its inner surface, and mottled with atheroma; on section of the vessel, this change was seen to engage the middle coat in large patches. The lining membrane had given way at one or two points near the valves, and had become detached to a slight extent from the middle coat.

CASE XLIX.—*Œdema of the Lower Limbs, followed by Gangrene; Enlargement of the Liver; Weak Impulse; Rapid and Irregular Action of the Heart; Hæmoptysis; Intermittent Musical Systolic Murmur at the Apex of the Heart; Venous and Arterial Throbbing in the Neck; Sudden Death. Enlargement of, and Fatty Accumulation upon, the Heart, and Fatty Degeneration of its Substance; Valves all virtually Sound; Cirrhosis of the Liver; Pulmonary Apoplexy.*

Richard B., aged sixty-nine years, a cabinet-maker, reputedly temperate, was admitted into the Mater Misericordiæ Hospital on the 16th of January, 1868. He was then generally anasarcaous; the lower extremities were greatly distended, and the fingers and toes were livid. The liver was uneven on the surface, and tender to pressure. The radial pulse was irregular, and so small and feeble that it was not calculable. The action of the heart was remarkably tumultuous and irregular, and varied in rate from 156 to 180 in the minute. There was strong venous and arterial pulsation in the neck. It was impossible to determine accurately the extent of precordial dulness. At the apex, and faintly at the base, but not transmitted in the course of the aorta, a remarkable musical murmur was heard with the first sound of the heart, but only with every fourth or fifth pulsation. The second sound was normal, as regarded quality, in both situations, but remarkably weak. The respiration was quick, 36 in the minute, and attended with dry râles all over the chest. Urine, sp. gr. 1·022, acid, and free from albumen, but loaded with lithates.

On the 18th he began to expectorate blood, which was occa-

sionally mixed with mucus. The pulse now became imperceptible at the wrist; the apex-murmur ceased to be audible; it was, however, again heard on the 21st, 22nd, and 23rd, after two days' silence. It was now much fainter than before, and of a squeaking character, not very dissimilar to the noise elicited by blowing through a reed.

On the 25th, a large bulla, the size of a crown-piece, appeared on the dorsum of the left foot. The feet were quite cold, yet the man complained of a burning sensation in them, and, in order to alleviate that sensation, he insisted on allowing them to hang out of bed.

On the evening of the 25th, having a call to the night-chair, he got out of bed, and a few minutes afterwards he was found dead in the sitting posture. He had been for some time previously incoherent.

A *post mortem* examination revealed the following condition of organs. The sinuses of the dura mater and the cerebral veins were remarkably injected. The lungs, with the exception of the base of the right, in which there was a mass of apoplectic extravasation, were healthy. The heart was enlarged, weighing eighteen ounces, with a considerable deposit of fat on its surface. The right chambers were dilated, and in a special degree the right ventricle; the tricuspid orifice was greatly enlarged. The left ventricle was scarcely dilated, but its walls were somewhat thickened. The valves, both mitral and aortic, were virtually normal, and apparently competent. At the root of the aortic valve some spicula could be felt by the finger, but they were manifestly incapable of impairing its efficiency, or of obstructing the current of exit; the mitral valve was of a deep yellow tint; the liver was enlarged, and in the early stage of cirrhosis; the kidneys were likewise enlarged, the cortex was thick and yellow, and the tubes contained oil dots in large numbers. The walls of both ventricles were in a state of fatty degeneration; those of the right in a more advanced stage than those of the left. There was a good deal of fatty deposit between the fibres.

The existence of mitral systolic murmur in this case, without valvular lesion, is, in my opinion, to be explained by the state of tissue-degeneration, and consequent yielding of the

walls of the left ventricle, as in some of those previously given. Its musical character was probably due to a flake of fibrin appended to the edge of the valve, and subsequently detached and broken up. But of this there is no positive evidence.

Of the interesting features presented by the preceding case, the following is still further illustrative.

CASE L.—Dyspnœa and Irregular Breathing ; Palpitation ; General Dropsy ; Hæmoptysis ; Enlargement of the Liver, and Slight Jaundice ; Failure of the Pulse ; Rapid and Irregular Action of the Heart ; Apex-Systolic Murmur ; Basic Double Murmur ; Gangrene of the Right Arm ; Death. Enlargement of, and Fatty Deposit upon, the Heart, and Fatty Degeneration of its Substance ; Dilatation and Hypertrophy of the Left Ventricle ; Dilatation, Atheromatous Change, and Inflammation of the Aorta ; Arteritis extending into the Carotids, Right Subclavian, and Axillary Arteries ; no Disease (virtually) of the Valves, and no Plugging of the Arteries ; Fatty State of Liver ; Congestion of Right Lung.

John W., aged sixty years, a gunsmith by trade, was admitted into the Mater Misericordiæ Hospital on the 9th October, 1867. He had been temperate, and had not had rheumatism. Up to six months previously, he had enjoyed uninterrupted good health; and then, for the first time, his respiration became quick on ascending a flight of stairs, and there was palpitation. A month later, swelling of the feet and legs appeared.

At the date of admittance, there was very considerable swelling of the feet, legs, and thighs; a rather remarkable superficial venous engorgement of the dorsal surfaces of the feet and lower part of the legs; and a slight jaundiced tint of the face and conjunctivæ. There was considerable effusion into the peritoneum; the liver was enlarged considerably: it extended nearly to the umbilicus. The pulse was 96, irregular, and intermittent. He occasionally sighed, and respiration was accelerated at intervals, even as he lay quietly in bed. The precordium was decidedly dull from the right margin of the sternum to an inch and a-half external to the nipple-line. In this latter situation, a distinct

bellows-murmur was heard replacing the first sound of the heart. It was audible as far as the left axilla, and likewise at the inferior angle of the left scapula. At the apex, the second sound was obscure, but still audible. Over midsternum, a systolic murmur, but less pronounced than that at the apex, was likewise audible, and here, also, a diastolic murmur was heard. There was visible pulsation of the radial and carotid, but not of the temporal arteries. Urine passed in moderate quantity, sp. gr. 1·015, acid, and with only a trace of albumen.

Up to the 19th, he continued to improve under a treatment consisting of tonics and diuretics; but towards the end of October, the penis and scrotum became swollen, an exceedingly unfavourable symptom in such a case. The penis was punctured, and on the following morning the discharge of serum had been so great, that it had filtered through the bed, on to the floor. Great relief was derived from this procedure, which was subsequently repeated. He soon began to sink; had repeated paroxysms of dyspnœa; blood was mixed with the sputa, and the lower part of the left side became dull on percussion.

On the 19th, a blush was observed on the left elbow.

On the 20th, this had extended to the shoulder and to the wrist, was attended with great tumefaction, and large vesicles had formed on the surface, which presented a lemon-yellow tint.

On the 21st, the tumefaction and redness, now of a dark hue, had extended to the right side of the chest and neck.

On the 22nd, the entire of the right upper extremity was tense, and large bullæ had formed on the elbow. The radial pulse was slow, only 72, whilst the heart beat at the rate of 156 in the minute. Cutaneous sensibility was impaired, as no pain was experienced on pressure.

He died on the morning of the 23rd.

The right upper extremity was in a state of incipient gangrene; the cuticle had been detached from the greater part of the surface. All the great serous cavities were full of liquid. The right lung was consolidated in its lower half, was dark and dry in section, and this part of the organ sank in water; the remainder of the right lung was cedematous. In the apex of the left lung,

there was a mass of calcified tubercle, and the surface of the lung corresponding to this was puckered. The heart was greatly enlarged, weighing, with one inch of the aorta and pulmonary artery attached, thirty-two and a-quarter ounces. It was fatty on the surface, and presented two "milk spots;" viz., one, remarkably white, on the anterior surface of the right ventricle about the centre; and another on the anterior surface of the left ventricle near the apex. The right chambers contained much dark coagulated blood. The tricuspid orifice was greatly dilated. The right ventricle was dilated and thickened in some degree, and its sinus, at the origin of the pulmonary artery, was dilated into a large pouch, presenting the appearance of a distinct chamber. Pulmonary artery dilated, but valves competent. The left chambers contained some dark clotted blood. The mitral valve was thickened, but not otherwise altered; it was competent to close the orifice, as proved by the water-test. The left ventricle was greatly thickened and dilated, and the left auricle dilated only. The walls of the left ventricle were nine-sixteenths of an inch thick at the apex, three-fourths of an inch at the middle, and two-fifths of an inch at the base. The cavity measured four and three-quarter inches from the root of the left segment of the mitral valve to the apex, and from the septum to the left wall, in the state of collapse, two and a-half inches.

Microscopically examined, with a power of 222 diameters, the muscular fibres of the left ventricle were found converted into strings of oil dots, which, after treatment with strong acetic acid, were seen arranged in unbroken linear series. No trace of transverse striation was anywhere visible. The aorta was dilated, atheromatous in patches, and of a deep scarlet tint throughout the arch. The aortic valves were healthy, but, owing to the dilated state of the vessel, they were inadequate. Water poured into the aorta flowed slowly into the ventricle through a small aperture in the axis of the opening. The deep scarlet tint of the aorta extended into both carotids, the right subclavian, and the axillary arteries; it was nowhere removable by washing. No embolus was found in any of the arteries of the right upper extremity, all of which were carefully examined.

The liver was enlarged, and contained much fat in the form of oil globules, both in the hepatic cells and interstices; it sank in water. The gall bladder was empty. The spleen was of normal size, and apparently healthy.

In relation to this case, the following remarks were made on the occasion of submitting the details, together with the diseased organs, to the notice of the Pathological Society of Dublin.* "The most interesting features in this case were, first, the existence of mitral regurgitant murmur without mitral valve-disease adequate to its production, as proved by dissection.

"The explanation of such an occurrence, which, in the absence of valvular lesion or inadequacy, must be attributed to yielding of the walls of the left ventricle during systole, was to be found in the state of tissue-degeneration of that chamber.

"Secondly. The existence of two murmurs at the base, without structural lesion of the aortic valve; namely, a systolic murmur produced by a rough state of the interior of the aorta immediately above the valves, where atheromatous change was most advanced; and a murmur of diastolic rhythm, produced by axial reflux, from incompetency of the valves to occlude the dilated orifice.

"Thirdly. The existence of aortitis, and arterial inflammation extending into the carotids and main artery of the right upper extremity, followed by gangrene of the right arm, of which it afforded the only rational explanation in the absence of arterial plugging.

"Fourthly. The existence of hypertrophy of the heart, to the extent of nearly threefold development, without valvular disease, and dependent upon disease of the aorta.

"Consecutive to hypertrophy there was fatty degeneration and softening of the ventricles, and mitral regurgitation by excentric yielding of the left ventricular walls, engorgement of the lungs, and general venous congestion and dropsy.

"Fifthly. Cardiac asthma or panting, and irregular respiration at intervals, associated with an atheromatous and dilated state of the aorta, of which I believe it to be pathognomonic.

* December 7th, 1867, *Proceedings*, vol. iii., part ii., new series, p. 233.

"Lastly, the existence of jaundice. In dropsy of cardiac origin, and connected with hepatic engorgement as its ostensible cause, I am disposed to regard jaundice as evidence of fatty disease of the liver, and inferentially, of the heart also."

CASE LI.—*Paroxysmal Dyspnœa; "Ascending and Descending" Respiration; Palpitation; Rapid Action of the Heart, and Failure of the Radial Pulse; Edema and Paralysis of the Left Arm; Cardiac Sounds Clear over Lower Sternum; Systolic Murmur; Death by Asthenia. Heart Enlarged, and Fatty on the Surface; Right Chambers Normal, and Structurally Sound; Left Ventricle Thickened, but not Dilated, except at Apex, where it was greatly Thinned; Fatty Degeneration of its Substance; Mitral and Aortic Valves Thickened and Inadequate; Aorta greatly Dilated, and slightly Atheromatous.*

James K., aged sixty-two years, a carpenter, temperate, admitted August 7th, 1869. For the last year he has suffered from slight palpitation and occasional shortness of breath, and eight weeks prior to admittance a beam of wood fell upon him, and gave him a great shock; since that date he has been more incommoded by palpitation and dyspnœa. A week since, whilst proceeding to his work one morning, he was suddenly seized with dyspnœa, felt as if he were dying, and was forced to return home. His breathing has been since constantly embarrassed, with occasional paroxysmal aggravations, which come on in the morning whilst he lies in bed; he then feels as if his breathing were about to cease. For the last few days he has spent much of his time at the open window, in order to obtain a free supply of air.

The face is slightly jaundiced. There is great dyspnœa. No œdema. Urine passed in small quantity, and frequently. Pulse arhythmically irregular, about 96 in the minute, but difficult to register owing to the existence of many abortive and scarcely perceptible pulsations. Heart's action foetal in character, arhythmically irregular, and varying from 168 to 192 in the minute; cardiac sounds morbidly clear and ringing over lower sternum, with a soft murmur accompanying the first sound. To have

Tinct. of digitalis, ℥x; Tinct. of perchloride of iron, ℥xx; Spirit of nitrous ether, ℥xl, every third hour; and at night, Hoffman's liquor, ℥xxx, with liquor of hydrochlor. of morphia, ℥xv, in camphor water.

14th. Has diarrhœa, with sickness of stomach. Medicine to be stopped, and grs. x of aromatic chalk powder to be given.

16th. Is much improved; can sleep in the horizontal posture; no diarrhœa. To have quinine and sulphuric ether, and to be allowed to get up.

25th. Confined to bed; radial pulse, 96, rate of 192; respiration "ascending and descending," *i.e.*, in the recumbent posture it became gradually quick and gasping, till it attained a rate of cardiac action 63 in the minute; this, which was its greatest rate, was reached in the course of about two minutes. It then as gradually subsided till it ceased altogether, remaining suspended for *twenty-two seconds*. At this rate, there would be little more than three respirations in the minute.

27th. No cardiac murmur to be heard. Pulse nearly suppressed in left arm, which is paralysed and swollen. Breathing, as last noted. It was particularly observed that during the period of apnœa there was no alteration of the heart's action, or of the radial pulse, in regard to rate or otherwise. He is only partially conscious, and is manifestly sinking.

Died quietly on the 30th.

The heart was enlarged, somewhat globular in figure, fatty on the surface, and blunted at the apex, which was formed entirely of the left ventricle. Attached to the apex there was a thick and tough shred of false membrane. The weight of the heart, with two inches of the aorta and one inch of the pulmonary artery attached, was twenty ounces. The aorta was enormously dilated, admitting readily the first three fingers introduced edgewise; it was slightly atheromatous. Two of the curtains of the aortic valve were thickened and nodulated on the free edge and lower surface, so that a slight dehiscence existed between them when approximated, permitting very slow passage of water from the aorta into the ventricle. The orifice of the aorta was not dilated. The right chambers were normal, and contained some decolorized clot, disposed in globular masses. The left auricle was much di-

lated. The mitral valves were thickened and agglutinated at the edges, so as to present a permanent opening of the figure of a button-hole, which, however, admitted the points of two fingers. The valves were incompetent. The left ventricle was thickened, but not dilated, except at the extreme apex, where it was so thin over a space equal to the point of the finger, that the thumb placed externally at a corresponding point, was readily felt through the ventricular wall. The muscular fibres of the left ventricle, examined microscopically, were found in an advanced state of fatty degeneration; but, by an oversight, I did not preserve a note of their precise appearance. There was some serous effusion in the right pleura. The abdomen was not examined. The structural integrity and normal condition generally of the right chambers of the heart, afforded an explanation of the clear and clicking character of the cardiac sounds over the lower sternum, and the absence of dropsy. The irregularity of action was a reflex of that which existed on the left side. The "ascending and descending" character of respiration, followed by a period of apnoea, was here exemplified in the highest degree. The aorta was greatly dilated. The close connexion between the respiratory phenomenon just mentioned, on the one hand, and the dilatation of the aorta and weak state of the left ventricle upon the other, I have already dwelt upon in the preceding pages.

Similar to the last case, in many respects, is the following.

CASE LII.—*Dyspnœa; Dizziness; Respiration of "Ascending and Descending" Rhythm; Rapid and Irregular Action of the Heart; no Œdema; Double Murmur at the Base of the Heart and in the Arch of the Aorta, the Systolic being transmitted into the Carotids; Diastolic Murmur, and Quasi-Presystolic Murmur at the Apex; Drowsiness; Death. Lungs Emphysematous and Congested; Heart Enlarged and in a state of Fatty Degeneration; Mitral Orifice slightly Narrowed, and Beaded with Vegetations of Lymph; Aortic Valves Thickened, and Incompetent; Aorta Dilated and Calcareous.*

James D., aged sixty years, a labourer, of temperate habits,

was admitted October 5th, 1869. His health was good up to three years previously; he then began to suffer from "gnawing" at the pit of the stomach, and weakness. Since then he has had dizziness in the head three times, obliging him to support himself against some fixed object, and giving him the sensation and appearance of being under the influence of alcohol. Three weeks ago his breathing began to be short. He never spat blood, and never suffered from rheumatism.

State on admission. There was no œdema. Pulse arhythmically irregular, and varying from 72 to 84. There were frequently two beats of the heart in rapid succession, both being represented in the radial pulse.

The carotid, tibial, brachial, and radial arteries pulsated visibly; the two latter being remarkably tortuous. The apex-pulsation of the heart was behind the sixth rib, half an inch outside the nipple-line; here, the first sound was prolonged, and associated with a murmur of quasi-presystolic rhythm. In this situation, likewise, a loud bellows-murmur took the place of the second sound, and was distinctly audible in the left axilla. The second sound was not intensified in the pulmonary artery.

At the base, a loud double bellows-murmur was audible, the diastolic being the louder; both were transmitted into the arch of the aorta, and the systolic likewise into the carotids. Both murmurs were transmitted some distance towards the apex, but were nearly lost at the seat of the apex-beat. To have Hoffman's anodyne, 3ss; and liquor of hydrochlorate of morphia, ℥x, at night; and tincture of perchloride of iron, and chloric ether, of each ℥x thrice daily.

For a fortnight the patient seemed to improve; the pulse was more steady, he slept better, took food in sufficient quantity, and the kidneys acted well. The medicine, having disagreed with the stomach, was then suspended, and replaced by quinine and sulphuric ether.

30th. Paroxysmal breathing, alternating with intervals of apnœa in the recumbent posture, were to-day for the first time observed; the transition from the one to the other being abrupt. Pulse very weak and intermitting; pupils dilated; patient somnolent and incoherent. The diastolic murmur at the apex was

very loud and blowing, replacing the second sound, and extending nearly to the end of the long pause. Its terminal portion was rough in quality, resembling in some degree the murmur of mitral contraction, but not continued *up to* the first sound. Double murmur faintly audible at the base, but very distinct throughout the arch of the aorta. Was moribund.

31st. He was unconscious. There were alternating periods of apnoea, and of paroxysmal and suspicious breathing, the former occupying twenty-four, and the latter twenty-six seconds. During these alternating changes of respiration, the pulse and the action of the heart underwent no change, and the transition from the state of apnoea to that of paroxysmal dyspnoea, was less abrupt than previously, being accomplished within a period of about one second.

He died at 8, P.M., and the body was examined on the following day.

The lungs were emphysematous and congested posteriorly. The heart was enlarged, weighing, with one and a-half inch of the aorta, and an inch of the pulmonary artery attached, nineteen ounces. The right ventricle was very thin, its internal and external layers being yellow, whilst the intermediate portion was of the natural hue. Under the microscope, the internal layers showed fibres in the stage of granular degeneration, and others in that of obsolescence. One of the latter measured only the 2,664th part of an inch in diameter.

From the upper part of the right ventricle a remarkable conical appendage of fat projected; it lay over the root of the aorta, and was about two inches long, and one inch broad at its base. The left ventricle was thickened; its walls measured half an inch at the base, and under the microscope its fibres exhibited some oil dots; but the transverse striæ were distinct.

The right auriculo-ventricular orifice was dilated, and both it and the right chambers were partially occupied by a large decolorized mass of fibrin. The tricuspid and pulmonary valves were healthy. Both auricles were normal; the mitral orifice was somewhat less than the ordinary size, admitting only the tips of two fingers. The mitral valves were competent, and in all respects normal, save that at the attached margin, and on the

auricular aspect, there was a circlet of warty vegetations of about the size of duck-shot; they were soft, partially transparent, and smooth on the surface. The aortic valves were slightly thickened, and shrivelled at the free edges; they admitted of axial reflux. In one of the sinuses of Valsalva, and partially attached to the wall of the vessel, there was a rugged mass of calcareous matter about the size of a pea. The orifice of the aorta was of normal size, but the arch was much dilated, admitting the tips of three fingers; on its internal surface there were visible a few yellow dots of incipient calcareous formation.

The only feature in this case which demands special notice, is the existence of diastolic apex-murmur not due to aneurism of the heart. It is, indeed, the only genuine example of the kind which has come under my notice.

I have, in several instances, met with a murmur of postdiastolic rhythm, *i.e.*, a murmur *appended* to the second sound; this is peculiar to extreme mitral stenosis. But a murmur *replacing* the second sound, and having its source at the apex, is so rare, that the dissection of a body in which its existence was verified clinically, possesses more than ordinary interest.

In considering the cause of this phenomenon, the basic murmurs must be excluded, because it is distinctly stated that they both fell short of the apex-point; whereas it was here and in the left axilla only, that the murmur in question was to be heard. The mitral orifice was in some degree contracted, both relatively to the left chambers, and absolutely; and on the auricular aspect the valve was beaded with a string of wart-like vegetations. To this twofold condition, conjoined with suppression of the second sound in the aorta, the substitutive murmur of aortic inadequacy not reaching the apex, I am disposed to attribute the mitral diastolic murmur.

In the great majority of examples of mitral stenosis, a second sound is heard at the apex; it there serves, though in a subordinate degree, to gauge the rhythm of the presystolic murmur, which usually succeeds it at a long interval. Occasionally, however, this murmur is broken up into two fragments; one appended to the second sound, and constituting a true postdiastolic murmur; and the other prefixed to the first sound, and repre-

senting the presystolic murmur which is alone heard in ordinary cases. Between these, there is an interval of silence. The fragments vary as to relative length in different cases; but usually the postdiastolic element exceeds the presystolic in this respect. It may even happen that the presystolic element is all but suppressed, it being distinctly audible only during periods of excitement of the heart; and at other times requiring strained attention in order to detect it (see cases of the girls Mary E. C., Mary F., and Thomas D., in "mitral stenosis"). If, in such a case, no second sound is audible at the apex, the diastolic gauge being thus wanting, the more prominent murmur, or, perhaps, the only one recognized, may be readily mistaken for a murmur of true diastolic rhythm. This I believe to have happened in the case under discussion. According to the view now stated, the murmur was in reality not diastolic, but postdiastolic in rhythm; having been misinterpreted, owing to the absence of the ordinary chronometric standard, namely, a second sound.

CASE LIII.—*Delirium Tremens; Quick and Feeble, but Regular Pulse; no Cardiac Impulse to be felt; Sounds Obscure, but unattended with Murmur; Sudden Death by Syncope. Congestion of the Lungs; Enlargement of the Heart; Dilatation of the Left Ventricle; Fatty Growth upon, and Granular Degeneration of, the Substance of the Heart.*

— F., aged thirty-four years, a large, fat, and flabby man of intemperate habits, was admitted into hospital, February 1st, 1870, in *delirium tremens*. For some weeks preceding January 27th, he had left off the use of stimulants; but on that day he drank freely, and shortly afterwards exhibited symptoms of *delirium tremens*.

When admitted he was tremulous and restless, and had not slept for three nights previously. The conjunctivæ were injected, and the pupils contracted, the tongue slightly coated, and the bowels confined. Pulse 110, soft and weak, but regular; skin cool.

On the following day it was found that he had not slept; he

had raved incessantly through the night, and several times got out of bed. Pulse remarkably weak and failing; no cardiac impulse to be felt, and sounds obscure, but free from murmur. To have gr. ss of morphia in two ounces of sherry every third hour, and plenty of liquid nutriment.

After the third draught he slept four hours, and on the morning of the third, whilst taking some beef-tea after awaking, he suddenly died of syncope.

The lungs were large in volume, and generally congested. The heart was covered with a thick layer of fat, and so soft, that when laid on the table it expanded by its own weight. It weighed nineteen and a-half ounces, and contained dark fluid blood, with one imperfect coagulum. The valves were all healthy. The left ventricle was dilated, but not thickened.

Examined microscopically, the muscular fibres exhibited transverse striation, with large blebs of fat floating over the field. A portion of the internal surface of the left ventricle, which was of a lighter colour than the remainder of the wall, exhibited fatty degeneration of the fibres in the granular stage with an absence of striation.

CASE LIV.—*General Debility; Attacks of Syncope; Visible Pulsation of Arteries; Double Murmur at Base of Heart, and Single Systolic Murmur in the Carotids; Diagnosis; Hypertrophy and Fatty Disease of Heart; Disease of the Aortic Valves; Dilatation and Roughening of the Aorta.*

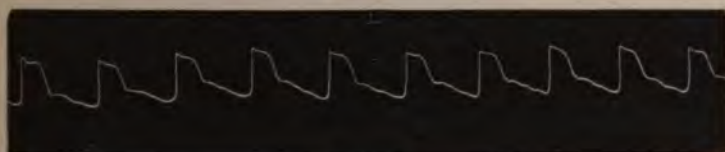
James D., a stout-built man, aged forty-nine years, a coachman by occupation, was admitted into hospital, June 27th, 1867. He had been intemperate, but never had rheumatism, or any other serious illness. About three weeks previously he began to lose power in all his limbs, but without experiencing any unusual sensations in them. There was wasting of the muscles, but no want of control over them. At the same time, he began to experience "a lightness" in his head, and his sight was obscured at intervals. A week previous to admission, whilst walking in the street, he felt dizzy, became pale, and would have fallen had

he not supported himself by holding on to a railing. This state lasted about a-quarter of an hour.

When admitted, he was listless and drowsy, and expressed himself slowly, but connectedly. Pupils normal and equal. Pulse 96, full, and equal in both wrists; visible pulsation in the radial and ulnar arteries. His aspect was fresh, and there was no cedema. Heart's impulse feeble and not extended. At midsternum a double murmur was heard, of which the diastolic element was the louder; it was transmitted in the course of the aorta, and at the second right costo-sternal articulation the systolic murmur was the louder. Here, likewise, it was coarse and jarring, and was alone transmitted into the carotids, which pulsated visibly but feebly. To have infusion of senega with carb. ammonia, grs. iij for the dose, thrice daily.

The annexed tracing of the pulse was taken by my friend Dr. Grimshaw on the 26th. On the 30th the man was discharged at his own request, much improved in general health, but with the physical signs unaltered.

FIG. XL.



James D.

Weak, but regular pulse; fatty degeneration, with obstruction and regurgitation at the aortic orifice.

There was no opportunity for testing the diagnosis in this case by *post mortem* examination; but I think there can be no doubt it was, in its main features, correct.

CASE LV.—*Anæmia, and Fatty Heart; Dilatation of the Aorta.*

Stephen McN., a porter in the butter market, aged thirty-seven years, temperate, presented himself as an extern patient at the Mater Misericordiæ Hospital, May 17th, 1867.

Twelve months previously he began to experience strange sensations in his head, and since that time he has been subject to giddiness, during which he has a tendency to fall down; has never had a fit, or become insensible. He is remarkably pale and waxy, and somewhat bloated in appearance, but not œdematous; the surface is cold, and he is very nervous. Both corneæ are somewhat opaque. Pulse 72, and weak. Respiration ordinarily 36 in the minute, but subject to remarkable variations of rate and character. Thus, the man being at rest, and the respiration 36, and inaudible to a bystander, it is liable to become quick, loud, and panting. In this state it would continue a few minutes, then subside to the previous rate, and resume its former character.

The surface of the chest is soft and puffy, but not œdematous; the heart's action is feeble; first sound scarcely audible; second sound much louder than the first, but less distinct than in the healthy organ; no murmur exists.

This man improved much under treatment with iron and quassia, and after a few visits he ceased to attend.

CASE LVI.—*Recurrent Vertigo; Orthopnoea; Chronic Cough; Rigidity and Tortuosity of Temporal Arteries; Urine slightly Albuminous, but sp. gr. 1.025; Pulse Regular; Hæmoptysis; Jugular Pulsation; Great Œdema and Tension of Lower Limbs, and Genitals; Erysipelatous Inflammation of latter; Left Apex-Systolic Murmur transmitted to Base; Death. Heart Enlarged; Right Chambers Dilated and Thinned, and containing a Band of Decolorized Fibrin, which passed through Tricuspid Orifice, and rendered Valve Incompetent; Tricuspid Opening Dilated; Left Chambers Dilated and Thickened; Dilatation being in excess in the Left Ventricle; Mitral Valve Sound and Competent; Aortic Valve also Healthy and Competent; Aorta Dilated above Orifice, and Atheromatous; Liver, Kidneys, and Lungs Congested; Advanced Fatty Degeneration of the Heart.*

Thomas O., aged sixty-nine years, a float driver, admitted

April 22nd, and placed under my care, May 1st, 1869, for pulmonary emphysema with chronic cough. He has had vertigo on exertion, but he never fainted. There is orthopnoea. Pulse, 108, regular; temporal arteries tortuous and rigid; impulse of heart heaving; both sounds masked and dull, but free from murmur; slight oedema of the feet.

After some weeks' residence, and under tonic treatment, he was discharged in improved health.

June 25th, re-admitted. Lower extremities and genitals are now enormously swollen, and on the left leg are some large red patches of an erysipelatous character. Urine, sp. gr. 1.025, and containing some albumen. Pulse 108, full and regular; cardiac impulse represented by an obscure and diffused heaving movement; no apex-pulsation to be detected, but below the nipple, and to the left of the nipple-line, a soft murmur, replacing the first sound, is audible; second sound weak, but normal. A transmitted systolic murmur is faintly audible at the base. Respiration 36, not embarrassed; slight expectoration of blood-stained mucus; jugular pulsation on right side. To have compound decoction of scoparium, 3viiss, with nitrous spirit of ether, 3iv; an ounce to be taken every second hour.

July 4th. Respiration has been greatly embarrassed during the last few days. Lower limbs swollen and tense, and the genitals in a state of erysipelatous inflammation. The pupils are contracted, and the patient is somnolent. At 12, A.M., he died quietly.

On the following day the body was examined.

The liver, spleen, and lungs were congested, but not otherwise altered. The heart weighed twenty-three ounces; it was soft, and, when laid on the table, it became expanded by its own weight.

The right ventricle was covered with a uniform layer of fat, two lines thick, and on its anterior surface, near the base, there was a large, white, irregular patch, caused by subserous thickening or deposit. The apex was blunted, and formed of the left ventricle exclusively. The aorta, near the heart, was greatly dilated, and atheromatous in patches; but the valves were sound and competent, as dilatation commenced only at the sinuses.

The right auricle and ventricle contained a ribbon-shaped

band of white fibrin; this band was attached by one extremity to the muscoli pectinati of the auricular appendix, and, passing through the auriculo-ventricular orifice, it fixed the left segment of the tricuspid valve to the anterior margin of the opening; whilst, by the other extremity, it was attached to the carneæ columnæ of the anterior wall of the ventricle. The right auricle was dilated, and the tricuspid orifice so much dilated that the four fingers and thumb passed through it together. The right ventricle was greatly dilated and thinned; the left auricle, dilated and thickened; mitral orifice normal, and mitral valves healthy and competent; left ventricle greatly dilated, and somewhat hypertrophied. The section of its walls presented the appearance of healthy structure, with the exception of the internal portion, including the fleshy columns, which were of a muddy yellow colour. Under the microscope, this portion of the wall presented the condition of granular degeneration, and here and there lines of oil dots were disposed in the length of the fibre.

In some portions of the field, fibres were presented in the stage of "obsolescence," having lost all appearance of definite structure and become greatly reduced in diameter. Transverse striæ were observable in the remaining portion of the wall of the left ventricle, but granular change had made some progress, and a few large oil dots were visible. The inner layer of the right ventricle consisted of fibres of a similar character to those composing the deep portion of the left ventricle; they exhibited granular change, with linear series of minute oil dots at intervals, and a few fibres in the state of obsolescence. One of these, where most reduced in diameter, measured the 1,776th part of an inch, and at an adjacent point, where, likewise, all appearance of definite structure had been abolished, the 1,110th part of an inch.

Jugular pulsation was noted nine days before death in this case, and extreme dyspnœa only a few days previous to that event. The inference seems, therefore, warranted, that, of the two causes adequate to the production of tricuspid incompetency, which the autopsy revealed, namely, extreme dilatation of the tricuspid orifice without proportionate expansion of the valves, and entanglement of a segment of the latter by a fibri-

nous thrombus, the former was that which actually gave rise to regurgitation, at least at this early date.

The existence of a murmur of mitral regurgitation, in the absence of lesion, or proportionate inadequacy of the mitral valve, as shown by the autopsy, is to be explained, as in many other examples of the same kind already given, only on the assumption of temporary yielding of the softened and dilated ventricle at the acme of systole, under the centrifugal pressure of the contained blood.

The next case, though observed at a much later date, is so germane to the preceding, that I deem this the most suitable place for its insertion.

CASE LVII.—*Chronic Bronchitis, Anasarca, and Ascites ; Arcus Senilis ; Rigidity, and Tortuosity of the Arteries ; Irregularity of Respiration ; Great Œdema and Sloughing of the Genitals ; Feeble Cardiac Impulse ; Faint and Fugacious Basic Systolic Murmur ; Death by Asthenia. Ascites ; Cirrhosis of the Liver, and Enlargement of the Spleen ; Slight Enlargement of the Kidneys ; Enlargement, and Advanced Fatty Degeneration of the Heart ; Roots of Aortic Valves Thickened and Rigid, but the Valves otherwise Sound, and Competent ; Mitral Valves Competent ; Right Chambers Dilated ; Left Auricle Dilated ; Left Ventricle Thickened, but not Dilated ; Arch of Aorta, from above the Sinuses of Valsalva, Dilated and highly Atheromatous, and its Lining Membrane Tinted Bright Red.*

John MacD., aged seventy years, a tailor, had drunk hard in early life, was admitted into the Mater Misericordiæ Hospital, February 6th, 1873. Six years previously, his breathing became oppressed, and obliged him to leave off work. One year later, he had rheumatism in the right shoulder ; and five weeks before admittance, his feet began to swell.

When admitted, he was in the following condition. Lower extremities and genitals much swollen ; face puffed, and surface generally pallid and pasty looking, except the feet, which were livid. Well marked arcus in both corneæ ; abdomen dis-

tended with liquid, but no turgescence of the superficial veins; slight cedema of the thoracic walls; cough with copious frothy expectoration; respiration averaging 30 in the minute, but exhibiting variation of rhythm. Thus, it was occasionally reduced to 24, and then gradually rose to 36, and at intervals of a few minutes it was entirely suspended for a few seconds. Pulse 108, regular, but very feeble, and visible; the right radial artery was rigid, and so tortuous, that about three inches above the wrist it passed to the outer and posterior surface of the radius, and after describing a long and gradual curve, resumed its position in front of that bone, and passed in the ordinary course beneath the extensor tendons of the thumb.

There was throbbing of the carotids. Precordial dulness was nearly abolished; no cardiac impulse to be felt; but at the lower end of the sternum the sounds of the heart were faintly heard. In this situation the first sound was feeble, toneless, and somewhat prolonged; the second sound weak, but rather sharp. A faint soft murmur was heard here, but, owing to the existence of loud bronchial râles, it was not possible to determine its rhythm satisfactorily. Bowels confined. Urine passed in moderate quantity, sp. gr. 1.020, and containing phosphates, but no albumen.

After a few days of treatment by means of stimulant expectorants, dry-cupping, and poultices to the chest, the poor man experienced relief from the bronchitis, and the murmur at lower sternum was determined to be systolic in rhythm; it was exceedingly faint, and not audible in the aorta or in the carotids.

In the further progress of this case, it was noticed that the pulse was occasionally irregular and intermittent; and, at the same time, the murmur was not audible; both events coinciding with an unfavourable change after a bad night's rest, or intolerance of food by the stomach. In the course of a day or two, when improvement had been effected by mild treatment, the murmur would again be heard.

On the 20th, the penis and scrotum being red and tense, were punctured; and on the following day they were passing into a state of gangrene. The features were now collapsed, and the pulse all but imperceptible.

On the 22nd he died of weakness; and on the following day the body was examined by my resident clinical clerk, Mr. Shanahan.

The abdomen was found distended with serum; the liver contracted, and as if seamed on the surface in several places. On section in these situations, the thickened capsule was seen to penetrate the organ. The parenchyma was of a light brick tint, and, under the microscope, the hepatic cells were seen to contain, each, several minute oil dots; free oil was likewise diffused over the field in moderate quantity.

The kidneys were slightly enlarged, weighing five and six ounces respectively. The cortex was thickened, light in colour, and, under the microscope, the tubes were seen full of granular *débris*, and many of them dilated and constricted at intervals; oil dots were likewise seen in the tubes, and free oil dispersed over the field of vision. Many of the tubes were thickened, and presented the concentric arrangement of fibres described and figured by Dr. George Johnson.*

Lungs congested and lacerable, the right being enlarged. The heart was soft and flabby, and expanded by its own weight when laid upon the table. It weighed thirteen ounces and six drachms.

Aorta dilated, dotted on the internal surface with yellow patches, and in the transverse portion, rough with small rigid plates projecting by their edges from the surface. This condition existed likewise in the roots of the three great branches arising from the arch. The lining membrane was of a deep crimson tint, not removable by washing. The dilatation of the aorta commenced above the sinuses of Valsalva, and did not implicate the orifice. The aortic valves were competent, and structurally healthy, but on the ventricular aspect the festoon-shaped roots projected as thick and rigid seams, and must have given rise to an eddy in the current of exit. Anterior segment of mitral valve, to the extent of one-half its depth from the attached margin, thick and opaque, but not rough or incompetent. Right chambers dilated, but not thinned; the outer surface being covered by a layer of fat one line thick. The right auricle contained a fibrinous thrombus. Tricuspid orifice not dilated;

* *British Medical Journal*, February 15th, 1873.

tricuspid valves sound. The muscular substance of the right ventricle was of a light fawn tint; and, under the microscope, the fibres exhibited oil dots irregularly dispersed, but crowded in most of them. In a few, the dots existed only in groups in the situation of the nuclei, the greater portion of the surface of these fibres having lost all appearance of definite structure. One of the fibres measured the 888th part of an inch; and another in the same field, but in the stage of obsolescence, only half that diameter, viz., the 1,776th part of an inch. (See engravings, Figs. XXXVIII. and XXXIX., pp. 614 and 615.) The left auricle was thickened, and somewhat dilated; the mitral orifice and valves (with the exception already mentioned) normal; the left ventricle was hypertrophied, but not dilated; its muscular substance was less light in colour than that of the right, but exhibited similar histological changes, except that of obsolescence. No striæ were anywhere visible in the structure of either ventricle.

CASE LVIII.—*Fatty Heart and Atheromatous Aorta ; Death.*

Rev. Mr. C., aged sixty-five years, tall and corpulent, consulted me on the 14th of July, 1869. He had had repeated attacks of rheumatic gout which had left his finger-joints enlarged and distorted; and, for the last few years, occasional vertigo with temporary loss of vision. Bowels constipated, and some hepatic tenderness. Respiratory sounds and pulmonary percussion resonance normal. Pulse arhythmically irregular and intermittent. Both cardiac sounds obscure below the nipple, and no distinct impulse or apex-beat perceptible. No murmur existed at the apex. At the right base, and for a distance of about two inches in the course of the aorta, a rough systolic murmur was audible, the second sound being clear. No murmur in the carotids, and no œdema. To have, every night, a pill consisting of blue pill, gr. j, and co. rhubarb pill and extract of taraxacum, of each *grs.* ij. During the day he was directed to take, three times, a table-spoonful of the following: Nitro-hydrochloric acid, 3ij; Liquid extract of taraxacum, 3iv; Infusion of cascarrilla, to 3xij. M.

July 20th. Pulse 96, regular; kidneys acting imperfectly.

To have, thrice daily, an ounce of the following; viz., Tincture of colchicum, 3iiss; Spirit of nitrous ether, 3iv; Camphor water, to 3xii. M. To continue pills, and paint enlarged finger-joints with liniment of iodine.

He returned home to a distant part of the country, and I lost sight of him. I subsequently learned that he died dropsical in January of the following year.

CASE LIX.—*Orthopnoea, Œdema, Hæmoptysis, Albuminuria; Enlargement of the Liver; Double Cardiac Impulse; First Sound Dull; Second Sound Clear and Sharp; no Murmur. Diagnosis: Renal Disease (probably Granular); Atheroma of the Aorta; Hypertrophy and Fatty Degeneration of the Heart.*

Rev. Mr. L., aged forty-eight years, consulted me, May 28th, 1872. Had had several attacks of rheumatism; for a short time preceding March 25th, he had cough, and on that night was suddenly attacked with severe cough and spitting of blood, accompanied by pain in the chest. Under medical advice he now took a few mercurial powders, which salivated him profusely. A week or two previously to his visit to me, his feet began to swell, and his breathing became much embarrassed. For the last few nights he was unable to lie down, and obtained only an occasional snatch of sleep in the stooping posture, resting his elbows on his knees. Within that period also he spat a little blood.

The following was his condition when I first examined him. The features were thin and shrunken; body plump, but remarkably pale and flabby; feet and legs greatly swollen. Liver enlarged. Urine acid, in sp. gr. 1·015, slightly albuminous, and containing fatty epithelial *débris*. Pulse 138, small and regular. Cardiac impulse double; apex-beat half an inch external to the line of the left nipple. First sound masked, and second sound clear and sharp. No cardiac murmur. To have aperient pills. To take, thrice daily, tincture of perchloride of iron, tincture of digitalis, and spirit of chloroform, of each ℥x, in an ounce of water; and at night a sedative draught of chloral hydrate (grs. xv), and liquor of opium (℥x), in an ounce of water.

May 30th. Slept very little last night, the greater part of which he passed in an arm-chair. Much cedema of feet, and great dyspnœa. Urine increased in quantity. To have mustard cataplasm to chest, and continue medicine.

June 1st. Much improved; quite free from oppression. Slept the greater part of last night in bed. Pulse 84, much more full, and quite regular; breathing tranquil. About three pints of urine were passed within the last eighteen hours. Some blood was passed from the bowels; but he has old hæmorrhoids. I now learned that the patient, having a dread of sedatives, *had not taken the chloral and opium draught till last night.*

4th. Swelling of the lower limbs has in a great degree subsided, and is now confined to the insteps and ankles. Urine averages about two quarts in the twenty-four hours. Can sleep soundly in the recumbent posture, and throughout the night, after his draught. No oppression or acceleration of breathing. Has only very slight and occasional cough, with simple mucous expectoration untinged with blood. Bowels regular; motions unaccompanied by needing, and free from blood. Pulse 84; strong, full, and regular, and presenting the characteristics peculiar to hypertrophy of the left ventricle. Appetite somewhat improved.

6th. Pulse quite regular. Cardiac impulse and sounds regular and distinct. He can now sleep with enjoyment in the recumbent posture, and take solid food; he walked to my house to-day, a distance of half a mile, without inconvenience. Has scarcely any cough, and swelling of feet has nearly disappeared. To have, thrice daily, Sulphate of quinia, gr. iss; Liquor of strychnia, ℥iv, in an ounce of water.

September 20th. Since last report he has had repeated bleeding from the bowels; the feet became again swollen; the right lung congested, and there was circumscribed pleuritis in the left mammary region. The pulse continued steady, and the patient was able to rest in the horizontal posture up to his death, which took place suddenly and quietly at 2, A.M., this morning.

The sudden break up of this gentleman's health after the injudicious administration of mercury deserves special notice. This I believe to have been the result of pre-existing renal

disease. In the combination of granular disease of the kidneys with consecutive hypertrophy and fatty degeneration of the heart, I have found even the smallest quantity of mercury inadmissible. Two grains of calomel have, to my knowledge, caused salivation and great debility in a case of this kind. The improvement in the condition of the patient under treatment with iron and digitalis, and a sedative of chloral and opium at night, likewise deserves notice. The kidneys acted vigorously under this treatment; the œdema all but disappeared; the heart acted with greater force; the breathing and the appetite were improved; sleep was enjoyed in the recumbent posture, and bodily strength was, in a great degree, restored. The improvement was, however, as in all such cases, only temporary. The body was not examined after death; but the diagnosis given in the title of the case, seems, in my judgment, fully warranted by the preceding history.

CASE LX.—Acute Articular Rheumatism; General Depression; Weak Pulse; Sudden Death. Fatty Degeneration of the Heart.

Alice M'C., aged about forty years, was admitted into the Mater Misericordiæ Hospital, December 28th, 1871. She suffered from acute articular rheumatism, which dated three days back. The heart was not engaged, and the only noteworthy symptoms exhibited were general depression, with a remarkable weakness of pulse but without irregularity or intermission.

She died quite suddenly on the 3rd of January, 1872, on making a slight movement in bed. The fibres of the heart were found in the so-called "glassy" stage of fatty change.

This case well illustrates the remark previously made as to the gravity of all acute inflammations, no matter how trivial in themselves, where fatty degeneration of the heart exists even in an early stage.

CASE LXI.—*General Dropsy and Venous Congestion ; Dyspnœa ; Slight Albuminuria ; Extension of Precordial Dulness ; Systolic Apex-Murmur ; Transmitted Systolic Basic-Murmur ; Death by Coma. Enlargement of Heart ; Mitral Stenosis ; Narrowing, and Capillary Injection of the Aorta ; Pulmonary Congestion and slight Emphysema ; Granular Degeneration of the Substance of the Heart.*

Mary A., aged twenty-eight years, admitted January 4th, 1873. Had rheumatism when twelve years old, and since then has been delicate, and for the last few years, easily put out of breath. Never spat blood. Three weeks ago the feet and legs began to swell.

When admitted she was suffering from general dropsy, accompanied with great dyspnœa. The lower limbs were livid and greatly swollen ; there was ascites, and the face was puffed, and of a purple tint ; the root of the neck was swollen, and the cervical veins were distended. Pulse scarcely perceptible at the wrists ; but, counted by the heart, it was 84, and regular. Precordial dulness much extended to the right, and the cardiac impulse strong, especially behind the lower sternum. The apex-beat was half an inch external to the nipple-line ; and here a remarkably loud systolic bellows-murmur was audible, but no second sound. At the base there was also a loud bellows-murmur of systolic rhythm, but less loud than the former, and manifestly transmitted from the apex. In the course of the ascending aorta, and over the front of the chest generally, this murmur was likewise audible, but faintly in proportion to the distance from the apex ; it was *not* transmitted into the carotids. At midsternum the second sound was very faint, but somewhat more distinct over the root of the pulmonary artery. Urine passed in small quantity ; it contained a trace of albumen, amorphous sediment of lithate of ammonia, and many octohedral crystals of oxalate of lime.

Under the use of active purgatives and digitalis with iron, she improved, and was able to obtain short sleep in the recumbent posture ; the face lost much of its previous lividity ; the excretion of urine was increased ; the radial pulse became perceptible, was

84, and regular. She was, however, restless at night; and on the 9th, a sedative draught, containing ℥xv of the sedative liquor of opium (Battley), was ordered. In the course of the night she became comatose, and died at 3, P.M. the following day despite the most active measures to resuscitate her, including electricity, strong coffee, brandy, hypodermic injection of atropine, and vigorous slapping on the palms and soles. This latter measure alone succeeded in rousing her for a short time; whilst the magneto-electric current, directed from the neck to the precordium, rather depressed the action of the heart.

The body was examined on the 11th January. There were several pints of serum in each pleura. The lungs were congested, and slightly emphysematous on the anterior edges, but otherwise healthy. The pericardium contained about eight ounces of serum. The heart was large, weighing thirteen and a-half ounces, and flaccid on the right side. The posterior surface of the right auricle was studded with granular lymph, the product of recent localized pericarditis; a firm band of lymph connected the anterior surface of the right ventricle, where an opaque patch existed, with the phrenic portion of the pericardium. The right auricle was greatly enlarged, and somewhat thickened, as was likewise the right ventricle; in the latter chamber a yellow flake of fibrin was found impacted between the right segment of the tricuspid valve and the wall of the ventricle. The tricuspid orifice was greatly dilated. The pulmonary artery was likewise dilated, but its valves were healthy and competent. The left auricle was greatly dilated and thickened, and the pulmonary veins dilated. The mitral orifice was reduced to the size of the point of the little finger; it was of a button-hole figure, and smooth and thick on the edges. The papillary muscles were not hypertrophied; but the tendinous chords were thickened, shortened, and fused together. On the ventricular surface, the segments of the mitral valve were seen to form by their union a nearly horizontal diaphragm, slightly bulging towards the ventricle, with a button-hole slit in its centre; the long diameter of this slit was directed from before and from the left, backwards and towards the right side. The left ventricle was somewhat thickened, but its cavity was nor-

mal. The aorta was reduced in size, and its lining membrane was of a deep crimson red colour, not removable by washing. The right and left anterior segments of the aortic valve were thick, opaque, and flaccid; on their ventricular surface, at some distance from the free margin, there were attached two or three nodules of rough lymph, of the size of a pin's head. The valves were, however, competent. Some dark clotted blood was found in the left auricle and ventricle. Under the microscope ($\times 222$), the substance of the right ventricle presented a typical example of the granular stage of fatty degeneration. The fibres had lost, nearly throughout, all appearance of transverse striation; they were pale, averaging the 1,110th part of an inch in diameter, and dotted over with dark points, which effected an arrangement, partly longitudinal and partly transverse, in relation to the fibres.*

There were four gallons of serum in the abdomen; there was slight adhesion of the convex surface of the liver to the diaphragm; the kidneys were of normal size, but the cortex was thick, opaque, and rather yellow.

For five days this poor woman was under my observation, and during that time I examined the heart daily; yet I did not detect the pathognomonic murmur of mitral narrowing, that of mitral reflux alone having been identified.

This is the only case of mitral obstruction which has come under my notice, in which a sufficient opportunity for stethoscopic exploration having been afforded, the patient being not actually moribund when examined, I have failed to detect a pre-systolic murmur and diagnose therefrom mitral narrowing, proved by dissection to have existed at the time.

I can explain the exception only by supposing that the contractile power of the left auricle was so impaired by effusion into the pericardium and pleural cavities, that it was incapable of propelling its contents with sufficient vigour to produce a murmur.

Fatal coma, in the main, no doubt, due to general serous effusion, was probably aggravated by the moderate dose of opium given.

* See Fig. XXXVII., p. 613.

That the basic systolic murmur was transmitted from the apex, I infer from the absence of murmur in the carotids, and from the manifest inadequacy of the lesion at the orifice of the aorta, to cause a murmur of efflux.

CASE LXII.—*Pulmonary Emphysema and Chronic Bronchitis; Recurrent Syncope; Irregularity of Breathing; Systolic Murmur at Lower Sternum; Sudden Death by Syncope. Enlargement of the Heart; Fatty Growth upon its Surface, and Fatty Degeneration of its Substance; Dilatation, Atheroma, and Roughening of the Aorta; Slight Opacity and Thickening of the Aortic Valves.*

Patrick M'E, aged fifty-three years, a gardener, admitted into the Mater Misericordiæ Hospital, January 20th, 1873. He was then suffering from cough and dyspnoea. Two years previously he was in hospital, and under my care, for pleuritis on the right side. A fortnight before the date of admittance, whilst walking in the street, he felt a "glow" all over his body, he became dizzy, and he would have fallen had he not held on by some support within his reach. Since then he has actually fallen in the street and lost consciousness, but has "come to" in the course of a few minutes.

When admitted, he exhibited no œdema, or visible pulsation. The pulse was 84, and scarcely to be felt. There was evidence of pulmonary emphysema with bronchitis. No cardiac impulse to be felt. Respiration varying in rate, even in the recumbent posture, and occasionally suspended for a few seconds.

At the lower third of the sternum, a soft bellows-murmur, regarded at the time as diastolic in rhythm, but really systolic, was heard; the difficulty of identification arose from the circumstance that the heart being in some degree overlain by the lungs, the cardiac sounds were audible only at distant intervals amid the discord of bronchitic râles. The diagnosis made was, pulmonary emphysema with bronchitis, enlarged and fatty heart, and disease at the orifice of the aorta. To have tincture of digitalis, tincture of perchloride of iron, and spirit of nitrous ether, of each ℥x, thrice daily; blister and a poultice to chest.

On the 24th he was much improved in regard to the pul-

monary affection, and now the murmur at the lower end of the sternum was readily identified as systolic in rhythm. No second sound was audible in this situation. During the succeeding two days no murmur could be heard; but on the 27th it was again audible, and recognized as systolic. On the latter date the patient was aphonic; pulse exceedingly weak; in the evening he died in one of his usual fits of syncope.

The body was examined next day with the following result:

The lungs were large, emphysematous, and congested posteriorly, and on the right side there was evidence of former pleuritis. Trachea dilated. There were a few ounces of serum in the pericardium. The heart was enlarged, weighing eighteen ounces, and covered at the base and over the right ventricle with a thick layer of fat. In the latter situation the fat extended to a depth of two lines, and somewhat more deeply at the apex. The right chambers were dilated, and full of dark clotted blood. Left auricle normal; left ventricle slightly dilated and thickened, except at the apex, where it was not more than three lines thick. The valves were all healthy, except those of the aorta, which were opaque and slightly thickened, but competent; they could not have caused a murmur of exit or of reflux. The aorta was greatly dilated from a few lines above the valves throughout the arch; it was rough, and fissured through its internal and middle coats in several situations; and about one inch above the valves, a layer of fibrin, as large as a sixpenny-piece, was firmly attached to one of these rough patches. The muscular fibre of both ventricles was in an early stage of fatty degeneration, exhibiting groups of oil dots in the situation of the nuclei. Elsewhere the fibres were smooth and pale, and in most situations devoid of all trace of striation; the latter appeared to a very limited extent in the fibres of the left ventricle only, which were of a redder tint than those of the right ventricle.

The murmur heard at the base of the heart was not of valvular origin; it was caused by the rough state of the interior of the aorta; and the great dilatation of this vessel, combined with the weak state of the heart, was the cause, at once, of the recurrent syncope, irregularity of breathing, and suppression of the second sound.

CASE LXIII.—*Vertigo, and great Debility ; Dyspnœa ; Weak and Visible Pulsation of the Radial Arteries ; Systolic Basic Murmur Transmitted into the Carotids ; Sharp and Clear Second Sound without Murmur ; Subsequent Suppression of Second Sound ; Acute Pericarditis ; Death. Emphysema and Engorgement of Lungs ; Hypertrophy and Granular Degeneration of Heart ; Villous Deposit of Lymph on the Pericardium and Heart ; Calcareous Transformation of the Aortic Valves ; Dilatation and Atheroma of the Aorta ; Liver, Kidneys, and Spleen Congested, but Structurally Sound.*

Patrick S., aged fifty-five years, a labourer, of temperate habits, was admitted into the Mater Misericordiæ Hospital on the 5th of February, 1872, suffering from cough, dyspnœa, and great debility. He stated that twenty years previously he had rheumatic fever, after which he was subject to winter cold and cough. Five weeks before admittance he began to experience dizziness ; and, three weeks later, great and general weakness. There was, at the date of his admittance, evidence of pulmonary emphysema and sub-acute bronchitis. The respirations were 24 in the minute ; the pulse was 72, soft and weak, but regular ; it was visible in the radial arteries. Precordial dulness was all but abolished ; there was no distinct cardiac impulse, which was represented by a diffused and heaving movement over the lower sternum. The apex was felt to pulsate faintly half an inch below the nipple, and in the nipple-line. At the base of the ensiform cartilage a loud systolic murmur was heard ; it was diffused over the entire front of the chest, and transmitted into the arteries of the neck, louder in the sitting than in the recumbent posture, and loudest at the root of the aorta and in the ascending portion of the arch. The second sound was sharp and clear.

Under a tonic and sustaining plan of treatment there was gradual improvement, and in the course of a few weeks the patient left hospital to resume his work. The cardiac signs, however, remained unaltered.

On the 27th of February, 1873, this poor man was again admitted under my care. He was then suffering from great oppression of breathing, orthopnœa, cough, and extreme weakness.

The sputa were copious and frothy, but uncoloured. Pulse feeble, small, and visible at the wrists. A soft systolic basic murmur existed, as in the first instance; but there was now no second sound to be heard, and no diastolic murmur. Under the use of digitalis and iron, with wine, and counter-irritation to the chest, he improved considerably, was able to lie down, and to sleep in the recumbent posture.

About the 6th of March he was attacked with acute pericarditis; a double and very loud friction-sound was audible all over the precordium, and not modified by respiration; effusion into the pericardium gradually ensued, and he died of asthenia on the 12th of March.

The lungs were found of large volume, emphysematous, congested posteriorly, enveloped in old and dense false membrane, and universally adherent to the chest-walls. The base of the left lung was in the first stage of pneumonia. The pericardium contained about eight ounces of sanguinolent serum, and its internal surface, both parietal and visceral, was studded throughout with villous lymph resembling the dorsum of a sheep's tongue. The heart was globular, enlarged, and, with the pericardium attached, weighed twenty-three and a-half ounces; its substance was fawn-coloured, and in the granular stage of fatty degeneration. The left ventricle was dilated in a slight degree, but greatly thickened, its walls near the base measuring three-quarters of an inch. The right ventricle was dilated and thickened. Both ventricles contained decolorized cylinders of fibrin, extending into the aorta and pulmonary artery respectively. The mitral valve was competent, and, with the exception of the attached margin of the antero-right segment, which was calcified to the extent of about two lines of its depth, perfectly sound. The aortic valves were rolled up in the direction of the aorta, and completely calcified; the posterior-left segment forming with the anterior mitral an uninterrupted calcareous plate. The aperture left by the disorganized aortic valves was in the axis of the opening, and admitted only the point of the little finger. Water poured into the aorta readily flowed into the ventricle through this aperture. The coronary arteries were sound. From above the valves the aorta was dilated throughout

the arch ; it was likewise in some degree rigid and patchy with atheroma, the latter condition extending into the roots of the great arteries of the neck. The liver was large, but otherwise healthy, as were likewise the kidneys and spleen.

I may mention that in January, 1872, the diagnosis made, as recorded in my notes of the case taken at the time, was hypertrophy, obstruction at the orifice of the aorta, and dilatation of that vessel. At no period during his residence in hospital was diastolic murmur audible.

Dr. Stokes has remarked upon the frequent absence of diastolic murmur in aortic patency, where fatty disease of the heart and aortic obstruction likewise exist. I can confirm this observation, and would suggest that the explanation may be found in the rigid and inelastic state of the aorta, which is usually associated with the twofold condition of fatty heart and thickened or calcified aortic valves. In this state, although reflux may take place, giving rise to feeble, collapsing, and visible pulsation of the superficial arteries, it is not accompanied with sufficient force of current to develop a murmur, owing to the want of elastic reaction in the aorta.

Finally, I would remark, that of the entire superficial surface of the heart, the anterior aspect of the apex was alone smooth and free from papillated deposit of lymph. Professor Law, many years ago, observed that this exemption of the apex is of frequent occurrence, owing to the impact of that portion of the heart's surface against the chest-wall in ventricular systole.

CASE LXIV.—*Renal Disease ; Hypertrophy and Fatty Degeneration of the Heart ; Gangrene of the Right Foot and Leg ; Death by Exhaustion.*

Rev. Mr. G., aged about sixty years, was admitted into hospital, May 3rd, 1873. He had been subject for the preceding year to vertigo and threatened syncope, and for the last three weeks there had been œdema of the lower extremities and genitals. Respiration was accompanied by moist râles all over the chest. The pulse was 96, weak, but regular ; precordial dulness extended ; no perceptible cardiac impulse ; first sound masked and faint, and second sound sharp and clear at base ; there were fits

of paroxysmal dyspnoea at distant intervals. Urine 1·020, and after ebullition exhibiting albumen to one-half its quantity.

About the 6th, it became necessary to puncture the feet and legs, in order to prevent the occurrence of inflammation from tension of the integuments. A copious flow of serum followed; nevertheless, two days later, a large bulla appeared above the internal maleolus of the left leg.

On the following day this burst, cedema subsided, and gangrene proceeded no further in that limb; the dorsum of the right foot now exhibited a livid patch, which soon extended to the toes. Large bullæ, filled with amber-coloured serum, now appeared about the ankle joint, and on the following day, along the leg as far as the calf, which was hot and tender, whilst the entire foot was livid, cold, and insensible. He complained of severe pain in the leg, and was troubled with hiccup, which was relieved by the exhibition, every second hour, of a pill composed of grs. ij of dried soda, and ℥j of creasote. He slept pretty soundly, and in the horizontal posture, but with occasional startings.

He died rather suddenly on Monday, May 12th, without having previously exhibited any marked increase of debility. The body was not examined.

In this, as in Case 46, I think the gangrene of the foot and leg was due to local arteritis and thrombosis.

In the form of degeneration known as "brown atrophy," first described by Rokitansky, the heart is wasted, and its muscular structure of a dark ochre tint. A yellow granular pigment is deposited in the muscular fibres, either uniformly, or in fine rows between the fibrillæ and around the nuclei. Whence these pigment particles proceed is not known. This kind of atrophy, which is always general according to Rindfleisch, is most frequently met with in senile marasmus, wasting from inanition, and in the tubercular and cancerous cachexiæ.*

Simple softening of the heart is a condition anatomically allied to fatty degeneration, but pathologically in no respect kindred to it. As in fatty degeneration, there is a change in colour and

* *Vide* also p. 588.

consistence; but the histological characteristics of that condition are wanting, the origin and association different, the development more rapid, the duration only temporary, and the prospect from treatment much more favourable. I propose to exclude from consideration here the so-called granular softening, which in no respect differs from the early stage of fatty degeneration; and likewise inflammatory softening, which, by a different process, leads to the same condition. By simple softening of the heart, I mean only the condition of that organ so often associated with essential fevers of the typhus type. In this connexion, Laennec described a condition of the heart characterized by a change of colour either to a violet or a dead-leaf hue, great softening and friability, and diffused over the entire heart or confined to the left ventricle. The cardiac impulse is very feeble, and, according to him, both sounds are indistinct, the first being occasionally suppressed.

Laennec vehemently denies the inflammatory origin of febrile softening of the heart insisted on by Bouillaud, and declares that it is peculiar to the grave forms of essential fever, which are characterized by intumescence of the face, a spongy condition of the gums and lips, a fuliginous deposit upon the tongue and teeth, an earthy colour of the skin, inflation of the abdomen, and fetid dejections from the bowels. The pulse, he says, is remarkably quick throughout the fever; and where it is continued at the same rate into convalescence, he would diagnose persistent softening of the heart; an error of judgment subsequently corrected by Dr. Stokes. Laennec further assumed the coexistence, in most cases, of what he described as "a gummy or fishy" state of the muscles of animal life.*

Zenker, who examined over one hundred bodies of those who had died of typhus at Dresden, in the epidemic of 1858-62, found the voluntary muscles, chiefly the adductors and the recti abdominis, in a state of degeneration in different degrees, and to a variable extent "no less constantly than the characteristic alteration of the mucous membrane of the intestine." He described two forms of degeneration of the voluntary muscles as witnessed by him in this connexion; namely, the "granular" and the

* *Auscultation Médiate*, tom. ii., p. 532.

"waxy." The former, as judged by his account of it, differs in no respect from the granular degeneration of the heart already described as constituting a stage of transition into veritable fatty degeneration. The granules are albuminous, and readily soluble in acetic acid. In the "waxy" form the fasciculi are swollen, in appearance homogeneous, and white and glistening like solid wax. This change is due to the deposit within the sarcolemma, which remains unaltered, of "a peculiar protein substance resulting from the transformation of the syntonin," and differing entirely from the amyloid. This he regards as the more grave form of the two, as it does not admit of the restoration of the normal structure of the muscle. It is likewise the more common of the two forms, having been found in 70 cases out of 79, and of this number, it was in the advanced stage in 36. Under both forms the muscles are brittle, and in many cases actually fissured across the fasciculi.*

I have not seen changes in the voluntary muscles corresponding to either of these conditions; they are denied by Stokes and other writers of the highest authority.

Laennec regards softening of the heart as essentially due to a disturbance of nutrition.

Louis, a few years later, noticed, very fully, softening of the heart as occurring in typhoid fever. Out of a total of 47 cases, the heart was more or less softened in 23 of those that died between the eighth and twenty-ninth days of illness; and in 15 out of this number, it was flabby, moulding itself like a wet cloth into any shape given to it. It was, moreover, easily torn, dry in section, not enlarged, and in colour it resembled the peel of an onion. Softening of the heart was found in only one-third of the cases which were fatal between the twentieth and thirty-second day of illness. No other muscular organ was affected, and the voluntary muscles were in a healthy condition. The walls of the heart were thin, and its chambers, in extreme cases, contained only a few drops of dark coagulated blood mixed with air. In nearly all, the lining membrane of the aorta was red, either in patches or uniformly; the change of colour extending to the middle coat; it was likewise thick and soft. Louis denies

* *Archives Générales de Médecine*, Aout et Septembre, 1865 (abstract).

the alleged inflammatory origin of this condition of the heart, regarding it as the result of a special blood-crisis.*

The next, and the most valuable contribution to our knowledge of this subject was made by Dr. Stokes.† According to this writer, softening of the heart, at least in Ireland, is peculiar to typhus fever. It is usually associated with a dark petechial eruption, sordes, and a peculiar odour from the body. The heart is livid and soft, especially the left side, pitting on pressure, and easily torn, and often infiltrated with "an adhesive gummy secretion." There is no trace of inflammation, and the valves are unaffected. The external one-eighth of the substance of the left ventricle is homogeneous and without the appearance of muscular structure, dark, and resembling in section the cortex of the kidney. These changes are, in a few cases, exhibited only in patches. The right ventricle and the septum ventriculorum are occasionally affected. He has not seen thinning of the parietes, nor a dry state of the cut surface, as described by Louis. He maintains that in the production of this remarkable change in the structure of the heart inflammation has no share, and adds, "All the anatomical and vital phenomena of this affection point to the opinion that it is an example of one of the special secondary lesions of typhus, like the infiltration of the mucous glands of the intestine, and capable of retrocession, without consequent disorganization."‡ The signs characteristic of this change in the heart commence about the sixth day of illness and cease about the fourteenth. They continue therefore, ordinarily, about eight days. The signs bear intrinsic evidence of debility of the heart, and are briefly as follows: Feebleness, or entire cessation of impulse, and partial or complete suppression of the first sound; coincidently with these modifications of the cardiac phenomena proper, the radial pulse is weak, but regular and rapid; the rate amounting, in many cases, to 150 in the minute. It occasionally happens, that whilst no

* *Recherches sur la Maladie connue sous le Nom de Gastro-Entérite*, 1829, tom. i., chapitre viii.

† *Dublin Medical Journal*, vol. xv., 1838; and *The Diseases of the Heart and Aorta*, 1854, p. 366.

‡ *Opus citat.*, p. 373.

impulse or first sound can be detected at the apex, both are perceptible over the right ventricle at the lower end of the sternum. Again, whilst no impulse is perceptible in the position of dorsal recumbency, a faint apex-beat may be occasionally felt in the normal position, or somewhat to the left, by causing the patient to turn over on the left side.

Dr. Stokes remarks that in some grave cases he has known the two sounds to be nearly identical in quality, and closely resembling a weak second sound, the action of the heart being at the same time very rapid. This he very appropriately designates "fœtal action." Under the free use of wine, and likewise in convalescence, the pulse usually becomes slow, and the first sound again audible; whilst the cutaneous circulation is more active, and the temperature of the extremities rises. These are signs of favourable augury, and may be regarded as evidence of the beneficial action of wine when they follow its administration.

When, on the contrary, between the eighth and the fourteenth day of illness, the action of the heart continues rapid despite an ample allowance of wine, the conclusion may be drawn that stimulants are not agreeing with the patient, or acting unfavourably. If the pulse continue rapid in convalescence, he would infer the presence of some acute inflammatory complication in the chest or abdomen, or phlegmasia dolens.*

The distinction between softened and simply weak heart rests upon the following characteristics: The latter is due to some constitutional debility of a very palpable character, to a shock, or violent revulsion of some kind. The signs are proportionately rapid in development, and, in the event of a favourable issue, equally prompt in disappearing. The signs of cardiac softening, on the contrary, progress up to a maximum point and then recede, occupying a period of several days; they appear with quick pulse, the impulse of the heart becoming less and less distinct, and failing first at the apex. There is usually, but not always, a corresponding loss of the first sound. In convalescence the pulse may descend to 30 in the minute, and again rise to the natural standard, at which it continues. The impulse is reestab-

* *Lectures on Fever*, 1874, p. 188-9.

lished before the first sound, and both are perceptible at the ensiform cartilage earlier than at the apex. The observations of Dr. Stokes, epitomized in the foregoing remarks, have advanced considerably the scientific therapeutics of fever. For a full discussion of the subject, which could not be entered upon here, the reader is referred to Dr. Stokes' writings already quoted.

Whether any change in the elementary structure of the heart takes place in simple softening, I am not in a position to declare from personal observation; but that such is not the case, is no more than a legitimate inference from the fact, that within a period of a few weeks complete restoration to health not unfrequently takes place in cases characterized by the above mentioned cardiac phenomena; the action and sounds of the heart resuming, at the same time, their normal characters.

Doctor Murchison, however, in the first edition of his great work on fever, published in 1862, affirmed that in typhus softening of the heart, the muscular structure of the organ is in a state of granular degeneration.

I have not met with the changes in the voluntary muscles described by Zenker; and from his allusion to the state of the intestinal mucous membrane, I conclude that the fever with which he found them associated was what in this country would be regarded as typhoid or enteric. Yet in this form of fever Louis declared that the voluntary muscles, as examined by him, were unaltered. It must, however, be remarked that the means for examination of structure at Louis' disposal were very defective. The subject needs further investigation.

Doctor Ormerod has studied this pathological change in connexion with delirium tremens. He says: "Of five dissections after death from simple uncomplicated delirium tremens, of which I have records, the heart is noticed to have been particularly soft and flabby in four, and in four to have presented numerous small ecchymoses beneath the investing or lining membrane. In one, the heart, though flabby, was not dilated like the rest, but small and loaded with fat. Examination by the microscope failed, however, to detect the slightest evidence of fatty degeneration of its structure."*

* *London Medical Gazette*, 1844, p. 792.

In delirium tremens I have occasionally found the heart acting with great feebleness and rapidity, the first sound being all but suppressed. Such a condition involves the utmost danger to life; even a slight effort on the part of the patient may cause syncope and sudden death: an accident which I once witnessed in the case of a professional man well known in Dublin. In such cases I should advise the use of digitalis and iron in large doses.

Fibroid transformation of the heart, incidentally noticed at p. 510, is most frequently the result of antecedent inflammation commencing in the pericardium or endocardium and thence extending into the substance of the organ. Inflammation leads to hypertrophy, by causing irritation and actual growth of the connective tissue of the heart, and proportionate absorption of the proper muscular structure. Fibroid change may, however, originate in inflammation of the connective tissue of the myocardium itself; but this is very rare in comparison with the first mentioned mode of origin. Sir W. Jenner maintains that congestion of the substance of the heart may give rise to a modified form of fibrous transformation if slow in occurrence, moderate in degree, and either permanent, or often repeated. In such circumstances, induration, roughening, and thickening of the walls of the heart by interstitial exudation of fibrin may take place; the walls would be firm, tough, and apparently homogeneous on section; they would not, though thin, collapse, and the fleshy columns would stand out. Hence, the cavities would be permanently dilated. The colour may be paler or darker than normal. Microscopically, the structure would be characterized by indistinctness of striation, and close aggregation of fibres, protein molecules being interspersed between them, and in their substance. These molecules are liable to fatty degeneration, and when this change has taken place the previously rigid structure is soft and brittle.*

I think the condition here indicated is due to sub-acute inflammation of the connective tissue of the heart, and differs only in degree from that about to be described.

Doctor H. C. Wood, of Philadelphia, declares, as the result of

* *Medico-Chirurgical Transactions*, vol. xliii., p. 199.

numerous experiments in the production of "thermic fever," a form of sun-stroke, which, according to him, does not require for its development actual exposure to the sun, that "excessive rigidity of the heart, by coagulation of its myosin, is pathognomonic of sun-stroke," and usually occurs directly after death.*

One of the earliest recorded examples of fibroid conversion of the heart was published by Dr. Fothergill.† A gentleman, aged sixty-three, short and full, and subject for three years to fits of angina pectoris, died suddenly in a paroxysm of anger. The body was examined by John Hunter, who reported as follows: "The heart, to external appearance, was sound; but, upon examination, I found that its substance was paler than common, more of a ligamentous consistence, and in many parts of the left ventricle it was become almost white and hard, having just the appearance of a beginning ossification."

Fibroid metamorphosis of the heart is a veritable "connective tissue-hypertrophy," or hyperplasia, and due, as Quain declares, to chronic interstitial inflammation. The inflammatory irritation to which it owes its origin may commence on the surface, external or internal, and thence extend by continuity of texture into the substance of the heart. Pericarditis and endocarditis are, therefore, not infrequent causes of hypertrophy of the connective tissue, or so-called "fibroid" degeneration of the heart.

When the pericardium is adherent, the heart, according to Quain, is generally enlarged, but the increase consists of connective tissue only. When atrophy of the heart coincides with adhesion of the pericardium, there is, he thinks, veritable cirrhosis of the heart: a change which he regards as due to the infiltration of a denser fibroid tissue.‡

I believe, however, that when the substance of the heart has become the seat of hypertrophy of the connective tissue, the volume of the organ may be taken as an index of the age of the hypertrophied structure. Where the heart is large it will be found still to consist in great part of muscular fibre, and the history of the case will show that the antecedent inflammation

* Prize Essay, reviewed in *Medical Times and Gazette*, October 19th, 1872.

† *Medical Observations and Inquiries*, vol. v., p. 252, 1774.

‡ Lumleian Lectures, *Lancet*, March 23rd, 1872.

has not been very remote. Where, on the other hand, atrophy of the heart has taken place, the muscular fibre has entirely, or all but entirely, disappeared; the remaining fibres, when any such are visible, being in the granular stage of fatty degeneration, as shown by Bristowe.*

Wilks reports a case in which one-half the muscular substance of the heart was replaced by fibrous tissue, which was in continuity with an adherent pericardium.†

Doctor Bence Jones has recorded a case strikingly illustrative of the causal connexion between surface inflammation of the heart and fibroid change of the subjacent structure. Under the endocardium covering the septum ventriculorum there was a layer of fibroid tissue "only where the endocardium had been inflamed." Mr. Gray, who examined this fibroid material, reports that it consisted "of granules, with nuclei and nucleated cells embedded in it."‡

In another case, Dr. Bristowe reports on a similar fibroid substance found in small detached masses in the substance of the heart, that it consisted "of fibro-plastic deposit, composed of large nucleated cells, granular cells, and nucleated spindle cells, with granular and oily matter, and pervaded by the muscular fibres."§

Rindfleisch urges that parietal endocarditis differs in its results from inflammation of the valves, in the circumstance that it leads to depression or pitting of the surface, thinning and bulging outwards of the wall, and thus to partial aneurism of the heart. The part so affected is small in extent, situate usually on the anterior wall of the left ventricle near the apex, and composed of tendinous structure invested by the pericardium. At the edges a few muscular fibres are still visible, but intermingled with the fibrous structure. This process of tendinous conversion must, in his opinion, be regarded "as a direct extension of the chronic inflammation of the endocardium to the sub-endocardial and intermuscular connective tissue."||

Doctor Lionel Beale regards fibrous transformation of muscle

* *Transactions of London Pathological Society*, vol. vi.

† *Ibid.*, vol. viii.

‡ *Ibid.*, vol. vii.

§ *Ibid.*

|| *Pathological Histology*, New Sydenham Society's edition, 1872, vol. i., p. 293.

as a veritable degeneration, arising either from old age or from long continued suspension of muscular contraction, as in cases of nerve-paralysis. "In old age," he says, "much of the muscular tissue is replaced by fibrous material." And again: "The contractile material of the muscle has degenerated into fibrous tissue." "Although in the muscles of the aged there is a relative excess of fibrous tissue, it is never, within the limits of health, present in such proportion as to constitute fibroid degeneration."*

Doctor Ormerod looks upon this change in the heart as one of conservative substitution designed to fortify the walls of an attenuated and weak heart. But where, as in Corvisart's case, the walls of the heart are actually thickened, this view would not be admissible; and that it is not a means ordinarily adopted to strengthen a simply weak heart, is proved by its absence in fatty softening.

I regard fibroid conversion of the heart, whether local or general, as in all cases the result of antecedent inflammation of the connective tissue of the organ. Most frequently the sub-endocardial, or sub-pericardial connective tissue is primarily affected, by extension from the adjacent serous surface, the connective tissue of the myocardium being subsequently involved through continuity of structure. The latter may, however, be primarily engaged. The subsequent changes are those of shrinking and condensation of the neoplastic material, with wasting and ultimate removal of the proper muscular structure by the disturbance of nutrition arising from extrinsic pressure.

Corvisart† gives a remarkable example of rigidity of the heart, caused, no doubt, by fibroid substitution: a problem impossible of solution at that time, owing to the want of microscopic appliances. The patient, a woman of fifty-five, came under his notice in the year 1800. The heart was large; the ventricles rigid and elastic, and, when compressed, sprang back on the removal of pressure to the state of dilatation, like an india-rubber bottle. The left ventricle, when percussed, sounded like a plate of horn. He remarks that this condition, when primary, precludes dilata-

* *Medical Times and Gazette*, March 20th, 1889.

† Hebb's translation, p. 146.

tion of the affected chambers; and that dilatation, when associated with it, is of anterior date. Quain confirms this observation.

Doctor Ormerod has met with examples of fibroid change so complete, that the heart, without having undergone change of colour, presented a resemblance to a leather flask, and was apparently incapable of contracting, save very imperfectly.*

The symptoms to which this change gives rise, even when partial aneurism of the heart results from it, are not characteristic, inasmuch as they have reference rather to the associated lesions of the aorta and coronary arteries, which are usually rigid and calcareous.

Angina pectoris is frequently a prominent symptom, as exemplified in Fothergill's case previously mentioned. The action of the heart and the pulse are irregular, but not constantly, or in all cases; the impulse is partially or entirely suppressed. There is occasionally palpitation, and the cardiac sounds are weak, but curt and sharp.† The "creaking" sounds, assumed theoretically to result from this condition of the heart, I have not heard. In one instance (Case 65) I have noted a regurgitant murmur from inadequacy of the tricuspid valve, consequent upon permanent dilatation of the right ventricle and fibroid transformation of the right papillary muscles; in another case, under the care of my colleague, Dr. Nixon, which he kindly invited me to examine, there was likewise a murmur of regurgitation both at the mitral and the tricuspid orifice. On examination of the body, this was found to be due to fibroid transformation of the inner portion of both ventricular walls and of the papillary muscles, one of which, in the left ventricle, was remarkably attenuated, rigid, and biventral.

In the dissecting room at the School of Medicine, Cecilia-street, a still better example of fibroid transformation of the heart was presented to me in the session of 1873-4, by Mr. Foley, one of the students engaged in dissection. It was obtained from the body of a male subject, aged about forty-five,

* *British Medical Journal*, 1862.

† Laennec declares, on theoretical grounds, that they are ringing in quality, and heard at a great distance; but such, in fact, is not the case.

regarding whose history or last illness unfortunately nothing whatever could be learned. The heart was somewhat enlarged, the mitral orifice was greatly contracted, and the mitral valves and chordæ tendineæ remarkably thick and rigid. The left papillary muscles were entirely converted into fibrous tissue, and the walls of the left ventricle, to the depth of two-thirds of their thickness, measured from the internal surface, were changed into dense fibroid tissue which was disposed in separate patches, and was continuous with the papillary muscles at their parietal attachment. The endocardium, in the left chambers, was universally thick and opaque.

Save in the early stages, when inflammatory changes are still in progress, nothing can be accomplished curatively. When the process of atrophy has advanced beyond the initiatory stages, the treatment should consist in measures to promote general nutrition and give tone and strength to the heart. Quinine, in combination with strychnia, and digitalis with iron, are the medicinal agents chiefly to be selected. Nutritious diet and alcoholic stimulants in moderate quantity should be allowed, and rapid movement and fatiguing exercise must be forbidden. The following case affords a good example of this variety of transformation.

CASE LXV.—*Pulmonary Emphysema with Recurrent Bronchitis ; Venous Congestion and General Dropsy ; Systolic Murmur at the Ensiform Cartilage and somewhat to the Left ; Death. Enlargement of the Heart ; Hypertrophy and Permanent Dilatation of the Right Ventricle, with Sclerosis of the Right Papillary Muscles, and consequent Inadequacy of the Tricuspid Valve ; Left Ventricle Normal ; Mitral Valve Opaque, but Adequate ; Aortic Valve Healthy.*

Mrs. S., aged thirty-nine years, and the mother of nine children, was admitted for the second time into the Mater Misericordiæ Hospital under my care, on the 7th of January, 1874. Just one year previously she was under treatment in hospital for bronchitis, to which she had been subject in winter for the preceding six or seven years. She was then cupped, and got well in three weeks.

When admitted for the second time she was livid ; the feet

were swollen; respiration was laboured, and expiration greatly prolonged; no pulse could be detected at the wrist. The heart pulsated regularly but feebly behind the xiphoid cartilage, and both in this situation and for a distance of an inch and a half to the left, a soft systolic murmur was heard. This murmur was confined within the limits just mentioned, and was accompanied by a faint first sound; it subsequently assumed a musical character for three or four days, and, at a later period, it was entirely suppressed for about two days. The lower limbs became greatly swollen and required to be punctured. The feet became erysipelatous and ultimately gangrenous. She derived great benefit from large doses of spirits of turpentine and tincture of digitalis; also from a night-draught containing chloral hydrate and bromide of potassium, of each grs. xx.

She died, worn out, on the 13th of February.

The heart weighed ten ounces. The right ventricle was permanently dilated and much thickened; its papillary muscles and inner stratum were rigid and firm, and, under the microscope, exhibited fibrous tissue pervading the muscular structure, in the form of broad bands, whilst the muscular fibres were reduced in size, and presented very faint striæ. The tricuspid valve was healthy in structure, but inadequate to cover the orifice, which was dilated proportionately to the ventricle. The left ventricle was normal. The mitral valve was remarkably opaque and somewhat thick, but pliant, and competent to close the orifice. The aortic valves were sound. The surface of the heart was smooth, but presented a large "milk spot" which occupied the usual situation on the anterior surface of the right ventricle. The latter chamber was occupied by clot and some flakes of fibrin. The pericardium contained about eight ounces of serum.

Metamorphosis of the walls of the heart, to a greater or less extent into *cartilage*, is the result of a more advanced stage of fibroid conversion. Professor R. Smith has recorded a good example of this condition; the apex of the left ventricle, and some of the columnæ corneæ had been converted into dense white cartilage.* When referring to cartilaginous alteration in con-

* *Dublin Medical Journal*, vol. ix.

nexion with hypertrophy (p. 510), I stated that I had not met with an example of the kind. Case XL (p. 592) subsequently came under my notice. In that instance the cartilaginous formation consisted of plates, about one and a-half inch in diameter, ovoid in outline, and about three lines thick; they were embedded in the superficial surface of the heart to which the pericardium closely adhered, and had their origin in antecedent pericarditis.

Cancer of the heart is, in the great majority of instances, secondary or metastatic. Rindfleisch asserts that it is never primary, and Peacock could find only two recorded examples. Most frequently the seat of the primary cancer has been the lungs, pleuræ, or anterior mediastinum; its form has been in most cases medullary, with or without melanotic pigmentation.

Doctor Peacock gives a list of 44 cases pretty fully reported by other observers, and 2 which had come under his personal notice.*

Laennec had seen 2 examples of encephaloid in the heart, and Recamier had mentioned to him 1 of scirrhus.

In a special memoir (*Revue Médicale*, 1824) Andral and Bayle refer to 3 cases which they had seen, and Bouillaud, in his first edition (1835), to 14 cases.

Dr. Peacock's 2 cases were briefly as follows:

Case 1. A young man, aged eighteen years, emaciated, had cough and hæmoptysis; action of heart weak and quick, but no murmur. Extensive infiltration of encephaloid cancer into both lungs and bronchial glands was found. The base of the heart was encircled by a ring of cancer one inch thick, and embedded in the false membrane of an adherent pericardium. The myocardium was unaffected. The liver, the spleen, the mesenteric and Peyer's glands, were cancerous.

Case 2. A young woman of nineteen years; six months ill with cough, hæmoptysis, dyspnoea, and wasting. The left lung was found extensively infiltrated with soft cancer. Two masses projected into the pericardium, penetrated the auricles, and pressed on the vena cava. There was some cancer in the mesenteric glands.

* *Proceedings of the London Pathological Society*, vol. xvi, p. 100.

Doctor Peacock classifies, under four heads, the cases of cancer of the heart included in his list.

1. Primary cancer of the heart, of which he could find recorded only 2 examples.
2. Heart affected in conjunction with other, and especially with adjacent organs; viz., lungs, pleuræ, mediastinal and bronchial glands: 5 cases.
3. Cancer spreading to the heart, by contiguity, from the mediastinal, cervical, and bronchial glands: 8 examples.
4. Cancer of heart secondary to that of some other organ. This is the most common form, and of it 22 examples are given. They were distributed as follows:

In 3 cases it was primary in the eye;

1 in the cheek and face;

1 „ lower lip;

2 „ breast and axillary glands;

1 „ radius;

1 „ ribs and pleura;

7 „ abdominal organs;

1 „ inguinal glands;

1 „ uterus and vagina;

1 „ labia and clitoris;

1 „ glans penis and inguinal glands;

1 „ testicle;

1 „ thigh.*

Doctor Risdon Bennett mentions a very remarkable case, communicated to him by Dr. Sutton, in which a primary scirrhus of the left female breast extended through an intercostal space, by a narrow pedicle, to the heart, which it extensively involved.†

Examples of malignant disease have been recorded by Drs. J. Sims,‡ R. Bright,§ Prescott Hewett, and E. L. Ormerod.||

Since the publication of Dr. Peacock's statistics, I find recorded 9 examples of cancer of the heart, which are summarized in the following Table:

* *Loco citat.*

† *Thoracic Growths*, 1872, p. 144.

‡ *Medico-Chirurgical Transactions*, vol. xviii.

§ *Ibid.*, vol. xxii.

|| *Ibid.*, vol. xxx.

TABLE VIII.—CANCER OF THE HEART.

No.	Author.	Age.	Sex.	Species of Cancer.	Primary.	Secondary.	Penetrated.	Where situated.
1	Dr. Flint	25	M.	Scirrhous	1	Enveloped heart.
2	Dr. Sutton	53	F.	Scirrhous	...	To scirrhous of left mamma	Right ventricle	In wall of left ventricle.
3	Mr. Bricheau	24	F.	Scirrhous	...	To scirrhous of ovary	Right ventricle and auricle	Over base of heart.
4	Dr. A. Clark	Melanotic	...	To melanotic cancer of nearly all the organs	—	—
5	Dr. Stokes	23	M.	Scirrhous and colloid	...	To scirrhous and colloid of posterior mediastinum, and nearly all the abdominal organs	...	Exterior of right auricle and ventricle.
6	Mr. Persé White	56	F.	Encephaloid	...	To encephaloid of right lung and posterior mediastinum	Pericardium	Exterior of heart.
7	Dr. Ed. Bennett	60	F.	Scirrhous	...	Anterior mediastinum and left lung	Right ventricle	Walls of aorta and pulmonary artery.
8	Dr. Hayden	29	M.	Scirrhous	...	To scirrhous of anterior mediastinum	Pulmonary artery	Embraced aorta and pulmonary artery.
9	Dr. Mayne	45	F.	Scirrhous	...	To scirrhous of anterior mediastinum and left lung	...	Pressing on superior vena cava.

The only example of carcinoma of the heart which I have seen, namely, that included in the preceding Table, was as follows :

CASE LXVI.—*Carcinoma of the Anterior Mediastinum, involving the Aorta, Cervical Vessels, and Pulmonary Artery; causing Suppression of Carotid Pulsation, Systolic Murmur in the Pulmonary Artery, and Displacement of the Heart Upwards and to the Left; engaging likewise the Root of the Right Lung, and Compressing the Right Branch of the Pulmonary Artery. Death by Asphyxia from Effusion into both Pleural Cavities.*

Andrew M., aged twenty-nine years, a cabinet-maker, formerly intemperate, but latterly reformed, was seen by me as an extern patient on the 25th July, 1871; he was supposed at that time to be suffering from thoracic aneurism. He gave the following account of his illness. Two months previously he got out of health, the first change noticed being a swelling at the root of the neck. He subsequently lost his voice at intervals, and a few days before I saw him, he spat some blood.

When examined by me he was chilled and weak, and could not lie comfortably on the right side. When he attempted to do so he was instantly obliged to turn over, owing to the tumultuous action of the heart which was thereby caused. His face was livid, and especially so the lips, lobes of the ears, and tip of the nose. The neck was tumid, the cervical veins greatly distended, and the upper part of the thorax œdematous and livid. The pulse was 120, very weak, but regular, and somewhat weaker on the right side than on the left. He had no difficulty in swallowing.

A general examination of the chest led to the following results. The entire surface was resonant on percussion, and respiration was audible on both sides at all points, save under the right clavicle, where there was a total absence of respiratory sound. At midsternum there was a harsh murmur, accompanying the first sound of the heart, which was diffused over the entire surface of the sternum; and over the lower end of the sternum a strong diffused impulse was felt, whilst no movement

was discoverable in the normal seat of cardiac impulse. Systolic murmur was likewise audible on both sides posteriorly, but louder on the right side, internally to the angle of the scapula, than on the left. No impulse was anywhere discoverable posteriorly.

He was admitted into hospital on the 20th September. There was then greater œdema and lividity of face, greater tumefaction of the neck, and distention of the cervical and superficial thoracic veins. There was likewise œdema, but not lividity, of the lower extremities and external genitals. The pulse was 120 and regular, and nearly, but equally, suppressed in both radial arteries. No pulsation was to be felt in the arteries of the neck; but in the carotids a very faint systolic sound was audible, showing that a feeble circulation was still carried on through it. There was no dysphagia. The heart was displaced vertically upwards to the left third intercostal space, where the apex was felt distinctly to pulsate. In this situation, and over the entire anterior surface of the chest, a systolic bellows-murmur was heard; but its point of greatest intensity was in the left second intercostal space, close to the sternum. The second sound was normal. The right front of the chest was comparatively dull; respiratory sound was nearly abolished on this side, especially over the lower two-thirds, and vocal fremitus was absent to the same extent; it was perceptible, however, beneath the clavicle. On the left side dulness likewise existed, except superiorly, and vocal fremitus was distinct only in the subclavicular region. Respiration was audible on both sides posteriorly, but less distinctly on the right, whilst comparative dulness existed on both sides.

Towards the end of September he became subject to occasional paroxysms of dyspnœa at night. The face, neck, and chest became tumid and in the last degree livid, and the right hand and arm much swollen, but decubitus was dexter.

He died quietly on the night of the 7th October; œdema having previously all but disappeared from the genitals and lower limbs.

In July, and the early part of September, I inclined to the view that aneurism existed; subsequent observation led me to

change my opinion, and for the following reasons. First, the dulness in front was not associated with a corresponding impulse. Second, there was *upward* displacement of the heart, and the murmur heard was most distinctly audible *above* the point of apex-pulsation. Third, pulsation in the cervical vessels was completely abolished on both sides: a circumstance scarcely compatible with the existence of aneurism in the situation indicated by the physical signs. Fourth, even if the latter supposition were admissible, an aneurism so situated could scarcely be unaccompanied by tracheal stridor and dysphagia. For all these reasons the alternative diagnosis, that of carcinoma, was made.

On dissection of the body both pleural cavities were found full of serum; the anterior mediastinum was entirely occupied by a scirrhus mass which extended to the ensiform cartilage, penetrated the pericardium, and surrounded the orifices of the aorta and pulmonary artery; it had completely embraced these vessels, and all but occluded the latter. It had displaced the heart upwards, backwards, and to the left, and projected by two nodular growths into the pulmonary artery a short distance above the valves, completely closing the right branch of that vessel, and so constricting the left branch that it barely admitted a director. The arch of the aorta, from an inch above the valves to the descending portion, was completely ensheathed in the solid scirrhus mass, and reduced to the size of an artery of the second order. Its lining membrane was rugose. The innominate and its branches, and the left carotid and subclavian, were likewise surrounded, and were reduced by compression to the size of the brachial artery. The heart was somewhat hypertrophied; it weighed eleven ounces; it was otherwise perfectly sound. Both lungs were considerably engorged, but structurally healthy with one exception; namely, the root of the right lung was contracted and corrugated, being involved in an extension of the scirrhus tumor from the anterior mediastinum. The œsophagus and trachea were not involved. The murmur heard in the left second intercostal space was a veritable systolic murmur in the pulmonary artery, due to the penetration and ingrowth of the cancerous mass.

The annexed engraving, from a coloured sketch taken shortly after death by Mr. Burnside, represents faithfully the condition of the heart and great arteries. Bristles are passed from the arch of the aorta through its primary branches which are embraced by the scirrhus mass. The right ventricle and pulmonary artery are laid open, and the nodular projections of the cancerous tumor into that vessel, by which it was all but completely closed, are exhibited.

The symptoms and signs proper to cancerous tumor implicating the heart are of a vague and indefinite character, having refer-

FIG. XLI.



Cancer of the heart and the anterior mediastinum.
Andrew M.

ence to excentric pressure upon the heart and great vessels, but not indicative of the specific nature of the detruing body. The differential diagnosis will lie between cancerous tumor, and aneurism of the primary portion of the aorta. Thus, displacement of the heart, and the signs of pressure upon the pulmonary artery or superior cava, may be due to either; as, likewise, dulness on percussion, superficial pulsation, and pain. But displacement by aneurism of the arch of the aorta is never *upwards*, and the signs of pressure are variable in degree and in direction, and more rapid in progress. Absorption of the osseous parietes of the thorax, bulging, and external tumor, are peculiar to aneurism, and positively exclusive of mediastinal cancer; and, finally, the pulsation of aneurism is heaving, like that of a second cardiac centre. Both are capable of projecting by absorption into the cavities of the heart and great vessels, and of producing identical auscultatory signs; but the collateral results of such ingrowth differ in the two cases. In the case of aneurism, the walls of the sac having been previously incorporated with those of the heart, pulmonary artery, or superior cava, communication with the interior implies irruption of its contents and a corresponding derangement of the general or pulmonary circulation; whereas, the ingrowth of cancer involves results having reference only to local obstruction and auscultatory signs. Cancer is slower in progress, more frequently associated with hæmoptysis and signs of pleural and pulmonary inflammation; usually, likewise, cancer is discoverable elsewhere. The history of aneurism is frequently connected with a definite occurrence, such as pain after a blow upon the chest, or after a strain; its progress is usually rapid and fitful, and unassociated with disease of other organs except atheromatous degeneration of the arteries.

Tubercular development in the heart has been described; this I have not seen. Todd doubts its occurrence, and Otto has not witnessed it. Miliary tubercle in the pericardium, and acute tubercular pericarditis in connexion with acute tuberculosis of one or both lungs, I have met with. Scrofulous or caseous masses in the substance of the heart have been witnessed by Elliotson* and by Todd.†

* *Lumleian Lectures*, p. 32. † *Cyclopædia of Anat. and Physiology*, vol. ii., p. 637.

Townsend describes an example of the same kind. A man aged sixty-four years, was subject for several years to cough and dyspnoea of a remittent character aggravated by damp weather, and, latterly, to paroxysms of dyspnoea threatening suffocation at night. On examination of the body after death, a large quantity of blood was found extravasated in the left lung. The pulmonary veins, especially those of the left side, were greatly dilated, and at the auricular extremity they were reduced to the size of a mole by the pressure of a large "solid mass of tuberculous matter."*

Hydatids have been found in the heart. The cysticercus has been seen in the human heart by Andral; it is often witnessed in that of "measly" pigs.

The late Mr. O'Ferrall exhibited before the Pathological Society of Dublin an example of the same kind. The patient was ill only three months. Tubercles were found in the lungs, and six or seven cysticerci in the septum ventriculorum. There was also chronic renal disease.

Doctor Peacock reported an example of hydatids in the heart, apparently primary, as none were found in the liver or in any other organ.†

I have not met with an example of hydatids in the heart. I should say the symptoms and signs would be most indefinite and misleading.

Syphiloma of the heart is very rare, and never met with except in subjects saturated with the syphilitic poison. It belongs to the category of "tertiary" symptoms.

Ricord gives examples of what he regards as syphilitic nodules in the substance of the heart; they were yellow, firm, and solid like cartilage.‡ Other organs, viz., the liver, lungs, spleen, kidneys, and brain, are usually affected in the same subjects.

Lancereaux gives two examples of syphilitic or gummatous myocarditis. The gummata were of the size of peas or cherries, and of a greyish or yellowish hue. The adjacent structure of

* *Dublin Journal*, vol. i. (old series), May, 1832.

† *London Pathological Society*, October 15th, 1872.

‡ *Clinique Iconograph. de l'Hôpital des Vénériens*.

the heart resembled tissue undergoing amyloid degeneration. According to Oppolzer, gummata may soften and burst into the chambers of the heart, thereby causing general infection.

Doctor John Morgan has published some interesting cases of the same kind. In one of these, that of a female, aged thirty-seven years, and the subject of chronic syphilis, several cream-coloured gummata were found in the substance of the left ventricle. In all these cases the heart was atrophied, the adult organ weighing in one instance only four ounces, and the impulse and the first sound were feeble. There was likewise, in some instances, an anæmic basic murmur; but no sign of a specific or pathognomonic character existed.*

According to Dr. Moxon, the elementary structure of syphiloma presents no characteristic feature, consisting only of imperfect and perishing corpuscles, not distinguishable from those of tubercular matter.†

Staff-Surgeon Welsh maintains that syphiloma of the aorta, in the form of so-called atheromatous degeneration, is, amongst soldiers, "the most frequent lesion occasioned by syphilis," and he would explain by this fact the prevalence of aneurism in the army. It was exhibited in the proportion of 57 per cent. of the forty examples of secondary syphilis recorded by him.‡

A case very suggestive of the causal relationship mentioned by Surgeon Welsh has been published by Dr. G. W. McNalty. This case is given in abstract at p. 736. But it must be remembered that syphilis is very common amongst civilians, those at least who frequent our hospitals, yet aneurism is not proportionately or remarkably prevalent amongst them; whilst, on the other hand, disease of the aorta seems to be a special appanage of the soldier, for which adequate causes relating to his dress and discipline may be adduced. It must be added that no distinction, based upon symptoms or morphological peculiarities of structure, has been established, or even advanced, between ordinary atheroma and so-called syphiloma of the arterial coats. I think, then, the allegation that atheroma is identical with syphi-

* *Dublin Quarterly Journal*, August, 1871.

† *Guy's Hospital Reports*, series iii., vol. xiii., 1867.

‡ *Army Blue Book*, 1870.

loma, and that the frequency of aneurism amongst soldiers is explained by the prevalence of syphilis amongst them, is not proven.

Myocarditis, or inflammation of the substance of the heart, may be presented in the *acute* or in the *chronic* form. In both, the investing or the lining membrane of the heart is at the same time engaged, and occasionally, both the one and the other. The possibility of parenchymatous inflammation of the heart, unaccompanied by pericarditis or endocarditis, has been actually called in question by respectable authorities.

Neither Corvisart nor Bouillaud had met with an example of inflammation of the substance of the heart uncomplicated with peri- or endocarditis.

Stokes barely admits the possibility of its occurrence in an isolated form, but he has not met with an example, nor have I.

It will not be difficult to understand why the isolation of the muscular substance of the heart in the inflammatory process should be so rare, if indeed it be possible, when due consideration is given to the fact, that on both its surfaces it is not only closely invested by serous membrane of extreme delicacy of structure and susceptibility to irritation, but that the deeper or areolar layer of this membrane is structurally continuous with its connective tissue. Having myself not witnessed an example of myocarditis in which the pericardium or the endocardium was not likewise in some degree involved, and having seen no well authenticated case of it on record, I feel bound to regard such a contingency as outside the domain of established pathology.

The occurrence of inflammation of the substance of the heart to a limited depth, in connexion with pericarditis and endocarditis, is, on the other hand, so far from being uncommon, that in my opinion it constitutes the rule; and I believe that between these cases and inflammation of the walls of the heart in their entire depth, there is only a difference of degree. As to extent, it is rarely general, being most frequently confined to one or other ventricle, and usually to the left.

Laennec was not satisfied of the existence of a single well authenticated case of general inflammation of the heart.* In-

* *Traité de l'Auscultation Médiate*, vol. ii., p. 554.

flammation, he urges, generally augments the colour and the density of structures; whereas, in the alleged cases of general cardiac inflammation the substance of the heart was discoloured and softened.

Corvisart and Bouillaud, however, give several examples, and Rindfleisch gives one, of diffuse or general inflammation of the heart, in the person of a man aged fifty-four years, who was the subject of secondary syphilis. The muscular structure was heightened in colour "with a dash of violet," and increased in density; the section resembling caoutchouc in firmness, and like it exhibiting a certain iridescence. There were spots of ecchymosis under the endocardium and the pericardium.*

Mr. Stanley published a very remarkable example of suppurative myocarditis, which, however, was associated with pericarditis, and, as I believe, consecutive to it. The subject, a boy of twelve years, and an inmate of Christ's Hospital, became feverish, and on the following day had pain in one of his knees and in his head, accompanied with delirium, and followed by dyspnoea and coma. Death took place on the fourth day of illness. There was no pain in the chest, and no symptom referable to the heart. The cerebral vessels were found congested; the abdominal organs and the lungs were healthy; the pericardium contained four or five ounces of turbid serum with flakes of coagulated lymph, and the surface of the pericardium generally was reticulated with lymph. The size of the heart was natural, and on section its structure was seen to be nearly black, the fibres being soft and compressible. Distant and numerous small collections of dark pus were found in the walls of both ventricles, those near the outer surface lifting the pericardium in a few places. The walls of the auricles were soft and dark, but did not present collections of pus. The endocardium and the valves were only vascular throughout.†

Doctor Thomas Salter furnished the particulars of a case of suppurative myocarditis confined to the left ventricle. Here, likewise, there was pericarditis, but only in the stage of vascular turgescence. A man aged fifty years had a paroxysm of pain

* *Opus citat.*, p. 275.

† *Medico-Chirurgical Transactions*, vol. vii., 1816.

in the region of the heart and dyspnoea after a walk ; several such attacks occurred within the succeeding four weeks, but always after exertion of some kind ; there was then continuous orthopnoea, and precordial distress of the most aggravated character ; the pulse was quick and very weak, but regular, and the sounds of the heart were very feeble. Severe pain now set in over the left front of the chest, from the clavicle downwards. Death took place by asthenia. The right pleural cavity was found to contain some serum ; the left lung was attached to the walls of the chest by recent false membrane, and was in the first stage of pneumonia ; the pericardium was generally vascular, but contained neither lymph nor serum ; the endocardium and the valves were normal. The substance of the left ventricle, except to the depth of a few lines on either surface, was soft and yellow, and its entire thickness was pervaded by small abscesses, from the size of a pin's head to that of a pea. The ascending aorta was atheromatous.*

Doctor J. Risdon Bennett quotes from the *Bulletin de l'Academ. Roy. de Médecine*, April 11th, 1843, a case of acute carditis engaging the left ventricle, and ending in abscess which burst into the pericardium. The case was published by M. Gintrac of Bordeaux. The patient was a man aged sixty-eight years, and subject to palpitation, which became very severe after a violent fit of passion, and was followed by orthopnoea and œdema of the lower limbs. The action of the heart was violent and irregular, and the sounds extensively diffused, but no murmur was audible. There were, however, loud ronchi. Pulse small and frequent, surface cold, and countenance expressive of anxiety. The pericardium was slightly reddened, and contained a turbid reddish fluid like a mixture of pus and bloody serum. The heart was large, and covered on the outer surface with a layer of concrete pus. Over the left ventricle the pericardium was opaque and easily detached, and here an aperture was found leading obliquely downwards into a large cavity corresponding to the lower portion of the left ventricle, which was separated from the upper part of the ventricle by a thick wall of unorganized lymph,

* *Medico-Chirurgical Transactions*, vol. xxii., January, 1839.

and containing a mixture of grumous blood and pus. It is impossible to say whether the pericarditis was primary or secondary in the foregoing case, although it was assumed to have been secondary to the irruption of pus into the pericardium. He quotes from the *Gazetta Medica di Milano*, January, 1844, another example of suppurative myocarditis with pericarditis. The author was Dr. Dubini; the patient, a female, was ill only four days, and complained of feverishness, dyspnoea, and a feeling of weight at the precordium. There was a double *bruit* at the base of the heart, rough and extended in the course of the aorta; subsequently the sounds were feeble, and the pulse failed. The muscular structure of the left ventricle was yellow and infiltrated with pus; the aortic valves were inadequate, and the apex of the heart was adherent to the pericardium. The writer refers to a third case of purulent infiltration of the heart which occurred in the same hospital four days later.*

Graves has given a case of inflammation of the heart ending in suppuration. This, he remarks, is very rare, because inflammation of the heart generally proves fatal before suppuration can take place. A man of fifty-five years, and of full habit, had been for some months suffering from cough, dyspnoea, and palpitation. Anasarca followed, with great pain and distress referred to the heart, and darting over the chest. The pulse was irregular. There was loud *bruit de soufflet* and *frémissement cataire*. The diagnosis of hypertrophy and dilatation of both ventricles was made. Death took place suddenly a few weeks subsequent to the date of the foregoing report. There was effusion into both pleuræ. The heart was enlarged, and there was general adhesion of the pericardium to the heart by means of long bands of lymph, easily broken down except at the apex, where they were strong and firm. There was an abscess in the wall of the left ventricle, large enough to contain two ounces of pus, lined by a membrane, and opening into the pericardium by a rent near the apex. Both ventricles were thick, and the valves generally unsound; those of the aorta were ossified.†

* *British and Foreign Medico-Chirurgical Review*, vol. xx., 1845.

† *Clinical Medicine*, second edition, 1864, p. 558.

Latham has published an example of general purulent infiltration of the heart, consecutive to inflammation, and fatal in two days.*

Laennec has credited Benevenius with having been the first to describe an abscess in the walls of the heart, and declares that he himself witnessed only a single example, which occurred in the body of a child of twelve years. The abscess, which was in the wall of the left ventricle, was large enough to contain an almond. He mentions another doubtful example, and affirms that there are no special symptoms indicative of abscess in the heart, adding that it may be wholly unattended with symptoms, as in the case given by Benevenius.†

Bonetus has recorded several examples of suppuration of the heart.‡

According to Raynaud myocarditis most frequently engages the left ventricle, and its posterior wall by preference. The external and the internal layers are, he asserts, oftener involved than the intermediate substance. He quotes Dittrich as holding that primary acute myocarditis occurs nine times out of ten in males, and most frequently in those under thirty years of age. It may arise from mechanical violence, or may complicate acute articular rheumatism, puerperal typhus, or exanthematous fever. It may likewise follow pyæmia. Mankopff witnessed it in an epidemic of cerebro-spinal meningitis.§ Da Costa asserts that there are no symptoms or signs proper to myocarditis; or, if such exist, they have not been separated from those of pericarditis or endocarditis, either of which is likewise usually present.|| And Flint declares that its presence is not determinable during life.¶

Hope is of opinion that this condition presents no peculiar sign; but he thinks "it *may* be the cause of the feeble, fluttering, irregular, intermittent action of the heart, with suffocative symptoms, when these phenomena cannot be accounted for by

* *London Medical Gazette*, vol. iii., p. 118.

† *Opus citat.*, p. 555.

‡ *Sepulchretum*.

§ *Nouveau Dictionnaire de Médecine et de Chirurg. Pratique*, 1868.

|| *Medical Diagnosis*, third edition, 1870, p. 360.

¶ *Diseases of the Heart*, second edition, 1870, p. 555.

the presence of fluid in the pericardium, or of polypous concretions from endocarditis."*

Walshe thinks that great weakness and a fluttering character of the pulse *may* be due to inflammatory softening of the left ventricle; but he inclines to attribute these effects to the implication of the cardiac nerves. He declares that no pain, and not even an uneasy sensation, exists at the precordium, unless where pericarditis is likewise present. Yet he adds that he has no experience of the affection.†

Stokes says that of myocarditis, as distinguished from pericarditis and endocarditis, we know nothing; but he admits that the disease may exist in an isolated form. Its pathological anatomy, he adds, is but little known, because paralysis precedes disorganization of muscle, and extensive paralysis of the heart is fatal. The most frequent cause of the latter affection is a very acute myocarditis merging into the chronic form.‡

From the preceding quotations and references, it will be apparent that, independently of the implication by contiguity of the superficial muscular strata of the heart in the ordinary course of pericarditis or endocarditis, inflammation of the muscular substance to such a depth as to constitute a veritable myocarditis, though comparatively rare, is by no means absolutely so. It is further manifest, that as to the existence of symptoms and signs positively diagnostic of myocarditis, the greater number of writers hold a negative opinion.

Nevertheless, I venture to advocate the affirmative view; I believe the diagnosis of myocarditis is quite practicable, irrespective of the accompanying inflammation of the investing or the lining membrane, inasmuch as there are both symptoms and signs proper to it. If it can be shown that amongst the ordinary symptoms and signs of pericarditis and endocarditis, as they are witnessed every day in our hospitals, rapid failure of the pulse, suppression of the impulse and sounds of the heart, extreme precordial anguish, respiratory distress, and a sense of impending suffocation, are not to be included unless where peri-

* *Opus citat.*, p. 197. The italics are mine.

† *Opus citat.*, p. 260.

‡ *Opus citat.*, p. 109.

cardial effusion, cardiac thrombosis, or pulmonary complication exists, I think the actual presence of such symptoms, in association with pericarditis or endocarditis, would justify the inference that the substance of the heart was implicated in the inflammatory process.

I think I may assume that the above mentioned phenomena do *not* belong to the ordinary serous phlegmasiæ of the heart, *per se*; further, that they are of not very unfrequent occurrence in connexion therewith, where none of the secondary causes just mentioned can be adduced in explanation of their presence; and, finally, that in cases fatal in an early stage, where none of these consecutive or collateral accidents have occurred, indubitable evidence of parenchymatous inflammation of the heart may be always readily detected.

The evidence upon which I rely as warranting the diagnosis of myocarditis is the following. Actual rheumatic arthritis with manifest implication of the heart, or exposure to cold quickly followed by pain in the region of the heart, and, probably, at the same time, in the limbs and in the head; delirium; virtual or absolute failure, with extreme rapidity of the pulse; pallor, coldness, and clamminess of the skin; anxious expression and collapse of the features, and tendency to deliquium; not unfrequently incomplete paralysis of one side of the body; orthopnoea and gasping respiration, with an urgent feeling of want of air, and frequent sighing; pain and great oppression at the precordium; fluttering and irregular action of the heart, with occasional intermissions; feeble or suppressed impulse; faint, foetal, and all but extinct sounds; area of precordial dulness not more than normal in extent, with or without the acoustic signs of pericarditis, of endocarditis, or of both.

The foregoing symptoms and signs are rarely all associated in the same case. Those which are essential are, pallor, coldness, and tendency to syncope; failure of the pulse; pain and oppression at the precordium; orthopnoea; gasping and suspirious respiration; feeble, fluttering, rapid, and irregular action of the heart; weak but sharp sounds, both resembling the second, or of a foetal character; with a history of recent and acute accession. The actual condition of the lungs and pleuræ, the area and degree

of precordial dulness, and the absence of chronic valvular lesion, must be duly considered, as precluding acute pulmonary congestion, effusion into the pleuræ or pericardium, and endocardial thrombosis.

The following case will serve to illustrate the foregoing remarks, although I was not afforded the opportunity of verifying the diagnosis. It is an example of the affection in an extreme and fatal form.

CASE LXVII.—*Myocarditis ; Left Hemiplegia ; Death on the sixth day of Illness.*

On Sunday morning, March 9th, 1873, in consultation with Drs. McVeagh and More Madden, I saw Mrs. P., aged about forty-two years, and a chronic invalid from uterine derangement. On the Tuesday preceding she had called at Dr. McVeagh's residence in a fainting condition. He had her removed to her home, and witnessed in the course of that day several paroxysms of aggravated cardiac asthma, accompanied with severe precordial pain. She complained of numbness in the left hand and foot, and suddenly lost the use of the left side of the body.

When I saw her, she was propped up in bed, pale, and gasping for breath. The surface was cold, and there was no pulse to be felt at the wrists. Precordial dulness was not extended, and there was no perceptible cardiac impulse. The heart was acting with slight irregularity, at the rate of 160 in the minute. The sounds were both clear, but remarkably faint. The characters of the heart's action were very similar to those of the foetus *in utero*.

Doctor McVeagh suspected endocarditis with cerebral embolism. She had been taking small doses of grey powder; the precordium had been blistered and subsequently dressed with mercurial ointment, and champagne was being freely administered. Digitalis was suggested by Dr. Madden, and approved of by me. It was accordingly given in \mathcal{M} v doses, with \mathcal{M} x of spirit of chloroform every second hour. Other treatment continued.

She died of asthenia on the following day, and no examination of the body was made.

Doctor McVeagh's diagnosis of primary endocarditis was, I have no doubt, correct. The absence of murmur, in the state of extreme debility of the heart in which the patient was examined by me, is not unusual, even in cases of chronic valvular lesion. I regard the case, then, as an example of primary acute inflammation of the endocardium, rapidly and fatally implicating the substance of the heart.

CASE LXVIII.—*Albuminuria ; Paroxysmal Dyspnœa and Mitral Systolic Murmur ; subsequent Development of Presystolic Murmur ; Hypertrophy of the Heart, and Consecutive Endo-Myocarditis ; Mitral Stenosis ; Death.*

The wife of a medical man residing in the country was brought to me by her husband, in May, 1873. She was pallid, and suffered from occasional palpitation and a feeling of uneasiness about the heart. There was sufficient evidence of hypertrophy of the heart. A bitter tonic with steel was directed to be taken, and an opiate plaster to be applied to the precordium.

On the 26th of February, 1874, I again saw this lady, in consultation with Dr. Denham and her husband. She had been confined, under Dr. Denham's care, three weeks previously, of her second child, which was living, and healthy. For some time before confinement the feet and legs had been swollen. The urine was 1·027 in sp. gr., dark brown, acid, and contained a good deal of albumen, some epithelial casts, broken epithelium, and blood corpuscles. She had, nevertheless, a favourable confinement. The œdema had in some degree subsided, and the urine assumed a more natural appearance, when she began to complain of great oppression about the heart, and occasional difficulty of breathing.

When I visited her she was pale and agitated. The feet and legs were remarkably anæmic, considerably swollen, and readily pitted on pressure; there was orthopnœa; the breathing was rapid, and the slightest effort, even that of speaking, brought on a paroxysm of dyspnœa, during which respiration became still more rapid; she gasped for breath, moaned deeply, flung her

arms about, and demanded to be lifted upright, and that air should be admitted; the chest heaved, the nares were dilated, and the face wore an expression of great fear and anxiety. This condition passed away after about two minutes; but the slightest disturbance or excitement, even that involved in applying the stethoscope to the precordium, brought it on again. The pulse was 120, and small, but regular. Precordial dulness was extended to the left. The action of the heart was tumultuous, but feeble. There was a slight "jogging" or double impulse, and the sounds of the heart were obscure, and scarcely audible. The stomach was intolerant of everything taken, and the bowels were confined. A laxative enema was administered, and digitalis, in doses of ℥_{xx} of the tincture, was tried; but the stomach immediately rejected it.

On the following day, a musical systolic murmur was audible at the apex. The patient having had no sleep for several nights, a sedative draught, consisting of Hoffman's anodyne and Battley's liquor of opium, of each ℥_{xx}, was prescribed as a hypnotic.

On the following day I learned that she had slept nearly the entire night in the horizontal position, and within the preceding twenty-four hours she had only one paroxysm of dyspnoea.

Up to the present date (March 4th, 1874) she has continued to take this draught at night, and with the same benefit. She is now taking quinine and spirit of nitrous ether. There is an increase in the quantity of urine passed; the cedema of the feet is less; the pulse has improved in volume, and is now only 96, and the chest may be examined without inconvenience.

There has been for the last few days only one paroxysm, which occurred on the evening of the 3rd of March, and was very severe. In the course of that evening also the patient was for some time incoherent. She can take and retain solid food, and sleep in the horizontal posture, but only on the right side. The breathing is short, and the cardiac signs remain as last described; subsequently, a well pronounced and harsh presystolic murmur became audible, and continued up to her death. This murmur could not have existed in May, 1873, and its suppression in February, 1874, is readily explained by the weakness of the patient.

I have no doubt as to the nature of the case, the most noteworthy feature in which is the great relief from the urgent symptoms of paroxysmal dyspnoea, cardiac oppression and palpitation, which was derived from the use of opium and Hoffman's anodyne ether.

She died on the 25th of May, with signs of effusion into all the serous cavities. The body was not examined.

Myocarditis may be fatal in the first stage, or that of active congestion, by paralysis of the heart and asystole; or it may lead to :

1. Abscess.
2. Purulent infiltration.
3. Ulceration.
4. Fibroid transformation.
5. Aneurism.

Abscess of the heart, according to Rindfleisch, consists of pus corpuscles, albuminoid molecules, oil globules, and the *débris* of disintegrated muscular fibres. It is generally surrounded by a very soft layer of reddish grey material; it may detach the endocardium, or force its way between the lamellæ of the auriculo-ventricular valves, it may burst into the pericardium and so cause rapid death, or likewise into one of the cavities, most frequently the left ventricle, and cause pyæmia or embolism in some portion of the systemic arterial circulation. It may lead to aneurism, by communicating with one of the chambers; and, finally, its contents may be inspissated and encysted, undergo caseous change, and be thus tolerated by the heart. The small and disseminated foci found in the substance and on the surface of the heart in connexion with pyæmia, puerperal fever, glanders, and other forms of blood-poisoning, consist of aggregations of *vibriones*, which ultimately penetrate the muscular fibres, and usurp the place of the sarcous elements.*

Doctor Moxon reports a case of pyæmia in a girl aged nine years, arising from suppurative periostitis of the thigh, cranium, and hand, in which multiple abscesses were found in the walls of the ventricles of the heart and in the kidneys; one of the

* *Opus citat.*, p. 275, *et sequent.*

former was on the point of bursting into the pericardium, which was inflamed and rough with lymph, and contained several ounces of turbid liquid. He adverts to the connexion between suppuration of the periosteum and that of the kidneys and heart, and remarks that it is so constant that he believes "when peritonitis, with pyæmic symptoms, is the cause of death, abscesses in the heart and kidneys may be expected to be present."*

Examples of both abscess and purulent infiltration are given in the preceding pages. I need not, therefore, further dwell upon that result of acute inflammation of the heart in this place. *Ulceration* is a less common consequence. The following is a good example from Dr. Stokes.

A youth of eighteen years contracted a violent pericarditis from sleeping on the damp grass while the body was still warm after active exercise; the disease proved fatal in a little over a week. His suffering from cardiac anguish was extreme. On examination of the body, general hæmorrhagic pericarditis was revealed. The heart was covered with layers of chocolate-coloured lymph, and exhibited several patches of superficial ulceration from two to three lines in diameter, and of equal depth, engaging the false membrane and penetrating into the substance of the softened heart. The muscular structure was dark, but less so towards the inner surface. He quotes from Testa an example of ulcerative endocarditis in the body of a lady of twenty-eight years of age. After death there was found acute endocarditis of the left ventricle, with ulceration extending far into the aorta, causing rupture of the latter into the pericardium and death by hæmorrhage. The heart was fatty.†

Morgagni witnessed several examples of ulceration of the heart, both external and internal;‡ so likewise did Bonetus.§

Laennec mentions an example of ulceration in an hypertrophied heart; the ulcer was one inch long, half an inch broad, and four lines deep. Death took place by rupture of the heart. There was, during life, no evidence of this formidable lesion. ||

* *Medical Times and Gazette*, September 28th, 1872.

† *Opus citat.*, p. 109, *et sequent.*

‡ *De Sedibus et Causis Morborum*.

§ *Sepulchretum*.

|| *Opus citat.*

Hope regards ulceration as the most common cause of rupture of the heart.

Walshe says there may be secondary ulceration of the heart, from pyæmia or phlebitis. He likewise alludes to veritable apoplexy by extravasation into the substance of the heart, as a consequence of softening.*

Myocarditis may become *chronic* and lead to fibroid transformation, to a greater or less extent, of the walls of the heart; a condition appropriately designated "cirrhosis" of that organ by Bristowe. This is usually partial, and localized near the apex or in the papillary muscles, and may result in shortening of these, contraction of the ostia, or annular constriction of the infundibulum of the right ventricle. The latter lesion is rare in the adult, but not so in the infant. Aneurism of the heart may likewise be a consequence of fibroid transformation of its walls, from localized chronic myocarditis.

In the *treatment* of myocarditis, the urgency of the symptoms, and the serious consequences in prospect, demand the most prompt and decisive measures. I would commence by the application of a dozen leeches over the precordium, and would repeat this measure on the following day, if relief from dyspnoea and cardiac oppression were not in the interim obtained and the pulse had not improved in volume and rhythm. A succession of light, warm poultices should be applied over the leech bites to encourage the flow of blood; or, if the weight of a poultice be objected to, a layer of cotton wadding or of spongio-piline may be substituted. After the abstraction of blood, the patient should be quickly brought under the influence of mercury; and the readiest mode of effecting this object is that recommended by Dr. Law, and known as his method, which consists in the administration of very small doses of calomel at short intervals. I usually give half a grain of calomel, with two grains of sugar or a grain of James' powder, every hour, and I find that twelve such doses usually suffice to produce mild mercurial action. Alcohol, in the form of wine or spirits, in very minute and oft repeated doses, is absolutely necessary to sustain the weak and failing heart. The quantity should not exceed a teaspoonful of

* *Opus citat.*, p. 260, *et sequent.*

whiskey or brandy, given with water, or the equivalent of wine, every half hour, lest it accumulate in the blood and irritate the inflamed structures; the endocardium and lining membrane of the aorta being, in many cases, at the same time inflamed. Opium is necessary to procure sleep. A grain of the watery extract, or ℥xx of Battley's sedative, may be given, and may be repeated in two hours if not efficacious. If pericarditis constitute a principal, or even an important element of the case, a large blister should be applied early over the region of the heart. Alkaline beverages, such as soda, Seltzer, or Carrara water, should constitute the drink. These may be given with milk or a small quantity of claret. In the stage of convalescence, quinine, strychnine, and iron should be given in combination, somewhat as follows: R Sulph. quiniæ, grs. xxiv; Sulph. ferr. granulat., grs. viii; Liquor. strychniæ (B. P.), ℥xl; Acid. sulphur. dil., ℥xx; Syrupi limon., ʒij; Aquæ font., q.s. ad ʒviij. St. coch. mag. ter in dies. The occurrence of a rigor, accompanied with vomiting, might be regarded as indicating the occurrence of suppuration, and the symptoms of pyæmia, supervening upon those of myocarditis, as evidence that pus had found entrance into the circulation. Very little is, however, known from actual observation, of the symptoms of this accident.

Aneurism of the heart was a term employed by Corvisart to designate a state of dilatation of the chambers, with enlargement of the heart. The term aneurism is not now so employed, "dilatation," with or without hypertrophy, supplying a terminology adequate to the expression of these various conditions. By "aneurism of the heart" is now understood a yielding and pouching of a limited portion of the wall of one of its chambers, by which a sac is formed, communicating with the interior of the heart, and usually, but not necessarily, projecting from its outer surface.

The causes which may give rise to aneurism of the heart, and in the order of their frequency, are the following:

1. Chronic endo-myocarditis, leading to fibroid transformation of a limited portion of the walls.
2. Ulcerative endocarditis.
3. Partial rupture of the wall on the internal surface, including the endocardium.

4. Bursting of an abscess into one of the chambers of the heart, and entrance of blood into the cavity of the abscess.
5. Discharge of an hydatid cyst into one of the chambers, and entrance of blood into the hydatid cyst.

Of 74 cases collected from various sources by Thurnam,* the left ventricle was the seat of aneurism in no less than 58. In these 58 cases there were 62 aneurisms, inasmuch as the disease was multiple in 4.

The composition of the sac-walls in the 62 aneurisms was as follows: In 15 it was composed of muscular fibres and the pericardium; in 4, of the endocardium and pericardium only; and in 25, of all the structures of the heart; hence he concludes the greater number is, in the first instance, "true." In a few, the walls of the sac had undergone cartilaginous or calcareous transformation. An adherent pericardium, which was present in 21 of these cases, affords the principal protection against rupture of the sac externally. This accident occurred in only 1 of the cases of adherent pericardium collected by Thurnam, and in this solitary instance the fatal extravasation took place into the left pleural cavity. The contents of the sac were composed of laminated fibrin in 23 instances, and in 17, of dark clotted blood.

As to the seat of aneurism, Breschet held that it was nearly, if not entirely, confined to the apex of the left ventricle.

Thurnam declares this to be too exclusive. It was located at the apex of the left ventricle in 27 out of the 58 cases already referred to; at the base of the left ventricle, in 21 of these cases; and at an intermediate portion of the ventricle in 15. The interventricular septum was the seat of the aneurism in 3 cases.

He has collected 9 examples of aneurism of the left auricle. Of these, 8 were examples of general dilatation of the sinus, and 1 only consisted of a circumscribed projection of the wall of the sinus, as large as a nut, and containing dense laminated fibrin. All 9 cases were connected with narrowing of the mitral orifice.

In reference to this last observation I would remark, that where mitral stenosis exists the usual condition of the left auricle is that of excentric or dilated hypertrophy.

He justly regards inflammation of the structures of the heart

* *Medico-Chirurgical Transactions*, vol. xxi., 1838.

as the most frequent cause of aneurism, by leading to fibroid transformation and pouching of the wall within a limited area. This is usually of rheumatic origin. Such is likewise the opinion of Rindfleisch. As to sex, Thurnam says that of 40 cases in which the sex is mentioned, 30 were males and 10 females; whereas, in arterial aneurism males are oftener affected than females, in the proportion of 8 to 1 according to Hodgson, and 11 to 1 according to Lisfranc. The size of the tumor varied from that of a hazel-nut to that of the ventricle.

The disease is most common between the ages of twenty and thirty years, and again, in advanced life; whereas, according to Sir A. Cooper and Bizot, arterial aneurism occurs most frequently between thirty and fifty years. Mechanical injury, violent fits of anger, forcible straining whilst the breath is retained, and such causes, may likewise give rise to the disease.

Flint gives 5 examples of aneurism of the heart from his own experience.* Of these, 3 were in persons advanced in years, and in 2 instances the age is not stated. Three were males, 1 was a female, and the sex of the fifth is not given. The left ventricle was the seat of the disease in all; the apex in 2, and the base in 3 instances. The size of the tumor did not exceed that of an English walnut. The walls of the sac were composed of the pericardium and endocardium in 2 cases, and of the pericardium and muscular substance in 1. They were calcareous in 1, and in the fifth the composition is not mentioned. In 2, the contents of the sac were laminated. In 1, the state of the heart as to volume was normal, and in 2 it was greatly enlarged; but in both of these, valvular disease was likewise present. The symptoms and signs were in all neutral, or non-suggestive of the lesion actually present.

In regard to diagnosis, Dr. Flint expresses himself as follows: "The existence of this lesion is hardly determinable with positiveness during life."

Thurnam says: "The diagnosis of aneurism of the heart must, in the present state of our knowledge, necessarily remain very doubtful. Indeed, it is not to be expected that a diagnosis will ever be effected without the aid of information to be derived

* *Opus citat.*, p. 117.

from acoustic and manual examination." The symptoms, as classified by him in the order of their frequency, are as follows: Dyspnoea, in 15 instances; precordial pain, 14; dropsy, 10; palpitation, 9; anxiety, 8; syncope or syncopal tendency, 3.

From the particulars of 51 cases collected by himself, Dr. Quain concludes that cardiac aneurism is almost equally distributed in the five decades from twenty to seventy years, inclusive. Of these cases, 1 occurred in a child of twelve years, and 1 in a person aged eighty-two years. He further concludes that the liability of females is to that of males as 1 : 3; a proportion identical with that given by Thurnam. As to the cause of the lesion: in 32 cases, of which he possessed the history, it arose from ulcerative endocarditis in 3 instances; from circumscribed fibroid transformation in 21; from bursting inwards of a cardiac abscess in 2; and from circumscribed fatty softening in 6.

In reference to situation, it is generally in the left ventricle; out of a total of 303 cases referred to by him, in which it was so situate, it was located at the apex in 138; at the base in 101; and intermediately between these points in 67. The duration is, he thinks, doubtful, because the diagnosis is not practicable. It is usually fatal by rupture, or by impeding the movements of the heart.*

Dr. Cragie gives a list of 20 cases, including 1 observed by himself; in this instance the aneurism was situated at the base of the septum ventriculorum, was large enough to contain a walnut, and projected into the right ventricle, which was thereby contracted.†

Mr. Hodgson has published an example of aneurism engaging the apex of the left ventricle, nearly as large as the ventricle itself, and filled with laminated fibrin like an ordinary arterial aneurism. The sac was very thin, and contained no visible muscular fibre. The apex is, in his judgment, the usual seat of this lesion; and fibroid transformation of the wall, and tubercular deposit, its most common cause.‡

Mr. Higham Hill gives the case of a female, aged sixty-five, and thin, who was suddenly seized with acute pain in the region

* Lumleian Lectures, *Lancet*, March, 1872.

† *Edinburgh Medical and Surgical Journal*, April, 1843.

‡ *Provincial Medical and Surgical Journal*, December 9th, 1843.

of the heart, accompanied with dyspnoea and vomiting. When seen by him, she was in a state of collapse, breathing with difficulty, and inclined to vomit. There was still pain in the region of the heart. The action of the heart was regular, but very weak. There was no murmur. She rallied under stimulant treatment, but died suddenly, twenty-four hours later, by hæmorrhage into the pericardium through a rent in the right auricle, which communicated with another rent two inches lower down on the internal surface of that chamber. The wall of the auricle between these two openings was dissected into two layers. There was general fatty degeneration of the heart, the left side being hypertrophied and the right side atrophied.* The absence of murmur in this case can be explained only by the situation of the aneurism, the force of contraction of the right auricle being inadequate to the production of a murmur.

Staff-Surgeon M'Nalty recently published an example of aneurism of the left ventricle, arising, as he supposes, from syphilitic deposition in, and consequent fatty degeneration of, the heart.† The patient was a soldier, aged twenty-eight years, of very intemperate habits, and repeatedly the subject of syphilis, for which he was treated in hospital no less than twelve different times. No suspicion of cardiac disease would seem to have been entertained. This man was found insensible in bed, and died soon afterwards. An aneurism of the size of the end of the thumb projected from the apex of the left ventricle. Its walls were very thin and devoid of muscular structure, and from its cavity, which communicated with the left ventricle, and was lined by laminated fibrin, a devious passage led beneath the visceral pericardium to the base of the ventricle, and here an opening had been formed into the sac of the pericardium, through which a fatal hæmorrhage, amounting to four ounces, had taken place. The muscular structure of the heart had undergone fatty degeneration; and throughout its substance, but chiefly in the walls of the left ventricle, a yellowish-white deposit, apparently "gummatous" was presented, partly in nodules and partly diffused. Examined microscopically, this deposit was found to

* *British Medical Journal*, April 12th, 1873.

† *Medical Times and Gazette*, June 14th, 1873.

consist mainly of ovoid cells in an obscurely fibrillated stroma. The aorta, above the valves and in the transverse portion, was atheromatous. Doctor M'Nalty is of opinion that the case was one of primary syphiloma of the heart, followed by fatty degeneration and aneurism of the left ventricle.

Inflammatory softening of the heart is, in my judgment, the most frequent cause of aneurism of its walls ; but I cannot agree in the opinion expressed by Dr. Milner Fothergill, that the "yielding and pouching of the wall take place during diastole."* I am satisfied it occurs during systole.

The *treatment* of aneurism of the heart should be conducted on the same principle as in dealing with aneurism of the aorta ; namely, mental and physical excitement should be strictly avoided, and lamination of the contents of the aneurism should be promoted by all suitable measures. No means are more efficacious for this purpose than iron, and nutritious diet ; alcoholic stimulants being strictly prohibited. Eminently unpromising though these cases be, they are not absolutely beyond hope ; as at least one example of cure by calcareous transformation of the sac, and solidification of its contents, has been recorded by Dr. Wilks.

In the two following cases are presented the only examples of cardiac aneurism which have come under my notice. The first, for the advantage of witnessing which I am indebted to Doctor Stokes, was not primarily cardiac ; and in the second, the diagnosis rests upon physical evidence exclusively, as, fortunately, an opportunity for testing it by *post mortem* examination has not been afforded.

CASE LXIX.—*Varicose Aneurism of the Heart.*

A man, aged thirty-one years, a strolling musician of very intemperate habits, was examined by me at the Meath Hospital, on the 4th, and again on the 6th of February, 1871, by the kind permission of Dr. Stokes, under whose care he was. Six years previously he had been treated by Dr. Hudson in that hospital for an affection of the heart, from which date, till seven weeks prior to his second admittance, on the 18th of January, 1871, he had enjoyed good health with the exception of slight dyspnoea.

* *The Heart and its Diseases*, 1872.

About that time, after hard drinking and exposure to severe cold, his breathing became more oppressed and his feet swollen. He was now, by the advice of a dispensary physician, put under the full influence of mercury, and not deriving benefit therefrom, he sought admittance for the second time into the Meath Hospital.

When I first visited the man his condition was the following: The face and neck were puffed and livid; the lower limbs congested and swollen; there was urgent dyspnoea, which was accompanied by constant teasing cough. The lungs were oedematous and the liver engorged. The pulse was 108, weak, and irregular. Precordial dulness was extended, and the cardiac impulse diffused. At midsternum, a strong fremitus was perceived by the hand, and here, likewise, a double "booming" murmur was heard; the first was systolic in rhythm, and the louder of the two; the second was diastolic, and both completely replaced the normal sounds of the heart. The poor man informed me, that for some weeks previously he had been himself greatly disturbed by these sounds, which he compared to those made by "a woman churning."

On the occasion of my second visit, two days later, the fremitus was not to be felt at midsternum; but, in this situation, the double murmur was still audible, although manifestly by transmission only, and much less distinct; whilst at the apex, which pulsated feebly in the fifth interspace and nipple-line, two murmurs of identical quality and rhythm, but much louder, were heard. I was informed by Mr. Murphy,* the efficient and accomplished clinical clerk, that previously to that day a very faint and soft blowing murmur was alone audible at the apex. No abnormal impulse was anywhere detected. Both murmurs were distinctly audible over the entire left front, but not in the left back, or anywhere on the right side of the chest. There was no dysphagia, and no difference in the respiratory sounds on the two sides. I made the presumptive diagnosis of aneurism of the primary portion of the aorta, opening into one of the ventricles of the heart.

The patient died a few days subsequently, and the details of the case, with the viscera, were laid before the Pathological Society, on the 18th February, 1871, by Dr. Stokes, from whose

* Now a distinguished officer in the Indian Medical Service.

report the following particulars are borrowed. "The aorta was greatly dilated, and its tunics much diseased. From the anterior wall of the vessel, close to the semilunar valves, there sprang an aneurism which had attained nearly the size of a tennis ball; it may be said to have arisen from one of the sinuses of Valsalva. In its posterior wall there was a rounded opening through which a goose-quill could be passed. This aperture established a communication between the aneurismal sac and the right side of the heart, for the opening conducted into the conus arteriosus of the right ventricle, immediately below one of the semilunar valves of the pulmonary artery. The sac, which did not contain coagula, lay within the pericardium." (*Vide* Figs. XLII. and XLIII.)

FIG. XLII.



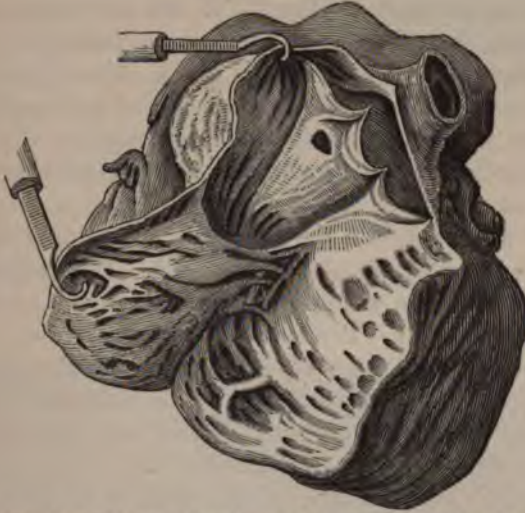
Aneurism of heart. Posterior view. (Case 69.)

At the request of Dr. Stokes, and with the permission of the President, I made the following remarks, as quoted from the *Proceedings* of the Society,* in reference to the grounds upon which the diagnosis of aneurism of the aorta, communicating with one of the ventricles of the heart, was based. "Dr. Hayden said he had but little to add to the very clear statement which Dr. Stokes had made with regard to this extremely interesting case. If he could correctly analyze his own impressions, and it was not

* *Proceedings*, vol. iv.

always easy to do so in retrospect, he might say that the impression of aneurism had existed in his mind from two sources. The first was that the murmur of exit was of a character which he had never heard in association with simple valvular disease. There had been a tendency of late to decry the independent value of the quality of a murmur, but he had always held that this was of the utmost importance in diagnosis. The murmur of exit at the base was of a *splashing* character, or such as would result from the sudden entrance of a body of liquid into a dense-walled and re-sounding cavity. Secondly, he inclined to the opinion that this murmur was not due to obstruction at the aortic orifice, mainly because it was not transmitted into the carotids. This seemed a point of some interest in differential diagnosis. He had not met with a single example of systolic murmur at the base, proved by dissection to have been due to disease of the aortic valves, in which the murmur was not distinctly transmitted into the carotids; this he regarded as a crucial test in doubtful cases. He had, however, repeatedly met with examples of basic systolic murmur not transmitted into the vessels of the neck; but in all such cases the cause of murmur resided in the aorta itself, and not in the valves. On the second day he examined this case, he found that the changes Dr. Stokes mentioned had taken place in the *interim*; namely, complete transfer of fremitus from the base to the apex of the heart. On the second day, likewise, the murmur that had existed at the base at the date of the first examination, was audible there only by transmission, and was now, according to his judgment, heard at the apex with undiminished intensity and unaltered character. This seemed to him a confirmation of his original view; for he could not, on any other assumption, comprehend the sudden and complete transfer of the thrill and murmur from the base to the apex of the heart. He could not, in short, reconcile these two facts with any other view save that of aneurism. Of course the diagnosis was rather presumptive than positive; that is to say, that the perfect confidence which one feels in dealing with familiar facts, was not experienced, owing to the rarity and eccentric character of the phenomena here presented. With the above reservation, the diagnosis was, great dilatation and advanced atheromatous transformation of the

FIG. XLIII.



Aneurism of heart. Interior of right ventricle. (Case 69.)

aorta, and aneurism of the aorta in the vicinity of the valves, establishing a communication with one of the ventricles of the heart."

I am indebted to Dr. Stokes for the use of the engravings from which the annexed impressions (Figs. XLII. and XLIII.), illustrative of the foregoing case, have been taken. Fig. XLII. represents a posterior view of the heart and aneurismal sac unopened; and Fig. XLIII. shows the interior of the right ventricle, with the aperture in the septum ventriculorum immediately below the right sigmoid valves, by which the aneurism had established a communication with the right ventricle.

By the kindness of my colleague, Dr. Nixon, under whose care the patient was admitted, I have had frequent opportunities of examining the following very interesting case. For the diagnosis I am alone responsible.

CASE LXX.—Double Apex-Murmur; Diastolic Apex-Thrill; Reduplication of Second Sound. Diagnosis: Aneurism of the Left Ventricle Located at the Apex, and Mitral Regurgitation.

James T., aged seventeen years, porter in a merchant's store,

was admitted into the Mater Misericordiæ Hospital, under Dr. Nixon, in the last week of May, 1873. He was delicate looking, with red hair, and very pale skin. Seven years previously he had an attack of rheumatic fever, in the course of which the heart was probably engaged, as he spoke of pain and distress experienced at the time in the precordial region. Six months prior to the date of admittance, he began to suffer from great weakness and shortness of breath.

When I examined him, the pulse was exceedingly weak and irregular, and not registerable at the wrist. The cardiac pulsations were 120 in the minute, irregular, unequal, and intermittent. Respiration 24. Precordial dulness somewhat extended to the left inferiorly. Apex-pulsation in the sixth inter-space, half-an-inch outside the nipple-line. The hand placed here experienced a "buzzing" thrill, synchronous with diastole, and accompanied by a movement of elevation and expansion over a space about the size of a shilling. At the point of apex-pulsation a double murmur was heard. The first was strictly systolic in time, soft, and blowing in quality. At a subsequent examination (11th June) it was of a musical character; it replaced the first sound, was loudest at the apex, and was likewise audible in the left axilla and back. The second murmur was postdiastolic, in the sense of accompanying the latter element of a double second sound; it was of a slightly booming character, and was strictly confined to the area of apex-pulsation. At the base an ill pronounced first sound was heard; also a double second sound, the latter element of which was remarkably loud and sharp in the pulmonary artery.

The boy is still (June 13th, 1873) under observation. The grounds upon which the diagnosis of aneurism of the left ventricle at the apex was made, are briefly the following. The diastolic murmur was of strictly apex-origin; an apex diastolic (not *postdiastolic*) murmur, is one of the rarest phenomena in cardiac acoustics, and can, in my judgment, only be due either to an abnormal communication between the aorta or pulmonary artery and one of the ventricles, as in Case 69, or to an aneurism springing from the wall of either ventricle. The former supposition was obviously excluded in this case, and the latter alone remained

to explain the phenomenon. This supposition was in my mind raised to conviction, by the existence at the apex of a diastolic impulse, of limited extent and expansile character, accompanied by a buzzing thrill, and by the elongation of the heart *without* aortic patency. That the systolic murmur was due to mitral regurgitation, I considered proven by its being audible in the axilla and left back, and by the intensification of the second sound in the pulmonary artery.

For the two following illustrations of the action of the heart and pulse in the preceding case, I am indebted to Dr. Nixon.

FIG. XLIV.



James T.

Cardiac tracing. Aneurism of left ventricle of heart. (Case 70.)

FIG. XLV.



James T.

Pulse tracing. Cardiac aneurism. (Case 70.)

*Wounds of the heart** may be considered under two heads; namely, those implicating the pericardium alone, and those engaging the heart.

Wounds of the pericardium are most serious; they are scarcely less fatal than wounds of the substance of the heart. They very rarely occur without implicating the heart itself, owing to the close adaptation of the investing membrane to the con-

* Though not belonging to the category of "Diseases of the Substance of the Heart," wounds of that organ will be most conveniently discussed in this chapter.

tained organ, equally in systole and diastole. Not only, however, *may* the pericardium be wounded without injury to the heart, but the heart may be wounded, even mortally, whilst the pericardium has sustained no apparent injury; having yielded before the impingent body, such as a bullet or other blunt missile or weapon, and been driven by it into the wound of the heart. At least four such examples are on record: one by Borellus; one by Professor Holmes of Montreal; one by Dr. Ward of Manchester, and a fourth by Dr. Fraser of the London Hospital. In the first case, which was described by Latour, a person was shot by a ball, and lived for three or four hours. The pericardium was found pushed into the wound in the heart, and, though filled with blood, was itself uninjured.*

Sir A. Cooper relates the following case of wound of the pericardium. A man was wounded with a reaping-hook which penetrated the pericardium; he survived about three weeks. A mixture of blood and pus was found in the pericardial sac.

Hennen relates the following. A man received a bayonet thrust through the diaphragm; he survived three months, and then died of pneumonia. The pericardium was penetrated, but the heart was uninjured.†

Doctor Parkes gives a singular case of this kind. A juggler, whilst exhibiting the trick of swallowing a sword two feet long, three inches broad, and round and blunt at the extremity, finding the weapon stopped low down in the œsophagus, violently pushed it onwards, and wounded the pericardium at its reflection from the right auricle. He immediately jumped from the ground, and fainted. Thirty minutes afterwards he complained of pain in the region of the heart and a systolic frottement was heard at the apex; this was double on the following day, and audible over the entire precordium. Percussion-sound was abnormally clear, no doubt from the presence of air in the pericardium. Death took place on the third day.‡

Wounds of the heart may be conveniently divided into *pene-*

* For the references to these cases, as well as for information of the utmost value on this subject generally, I am indebted to the able and exhaustive article of Mr. J. F. West (*St. Bartholomew's Hospital Reports*, vol. i., 1870).

† *Chelius' Surgery*, vol. i., p. 454.

‡ *Transactions of the Pathological Society of London*, vol. iii., p. 41.

trating and *non-penetrating*; and again, each of these classes may or may not be associated with *transfixion* of the heart.

Wounds of the heart, of whatever kind, may be *immediately* or within a few minutes, fatal from hæmorrhage. They may be *proximately* fatal, on reaction supervening, by dislodgment of a clot from the wound, or by pressure upon the heart from a coagulum formed in the pericardium, the result of a gradual oozing of blood from the wound; or *remotely*, by inflammation of the pericardium or the heart, or by the structural alterations resulting therefrom.

Fatal primary hæmorrhage usually proceeds from the chambers of the heart; but, when fatal at a later period, the bleeding is most frequently derived from a wounded vessel in the walls of the heart or chest.

Hippocrates declared that wounds of the heart were always fatal; whilst Galen held the modified opinion that penetrating wounds alone were instantly mortal, especially those of the left ventricle.

Ambrose Paré was the first who recorded an authentic case of wound of the heart; in that instance the wound was the result of a sword-thrust, and was large enough to admit the finger.

Muler recorded an example of wound of the right ventricle, the subject surviving sixteen days.

Job à Meeckren recorded the first case of traumatic pericarditis; and Morgagni was the first who showed that hæmorrhage into the pericardium was fatal by impeding the motions of the heart.*

The right ventricle, from its situation, is the part most frequently wounded. Next in frequency the left ventricle. The auricles, owing to the protection afforded them by the sternum, are least frequently the subjects of mechanical injury; but wounds of them are always penetrating, and yield the most copious and rapidly fatal hæmorrhage, owing to the extreme thinness of their walls.

Out of 121 cases collected by Jamain, the right ventricle was

* Raynaud, *Nouveau Diction. de Médecine, et de Chirurgie Pratique*, vol. viii., article "Cœur." Also Jamain, "Thèse de Concours pour l'Aggrégation," *Sur les Plaies du Cœur*.

wounded in 43 instances; and of those collected by Olivier, 61 in number, it was the part injured in 23 instances. Wounds of this chamber are nearly always produced by penetrating wounds of the chest; whilst those of the left ventricle have occasionally resulted from the passage of foreign bodies from the œsophagus or the stomach.

Wounds of the pleuræ and lungs constitute the most frequent, and likewise the most formidable, complication of wounds of the heart. They are almost invariably fatal when complicating wounds of the heart of whatever kind. Penetrating wounds of the lung may be readily diagnosed by the escape of bubbles of air from the external wound, and by the occurrence of hæmoptysis. Wounds of the heart, however, do not admit of such ready or facile diagnosis. When instantly fatal, unless the superficial wound be directly over the heart, it will be impossible to affirm with confidence that death was caused by injury to that organ. When death follows at a more remote period, the difficulty of positive diagnosis will not be so great; because time will have been allowed for the development of physical signs, and for the due estimation of the symptoms and other evidence actually present.

The symptoms and signs indicative of wound of the heart are the following. Faintness, or actual syncope; terrified expression of face; pallor; quick and feeble pulse; fluttering action, and sometimes pain in the region of the heart; sighing respiration; cold surface; weak voice and great thirst; and occasional nausea and vomiting. The sounds of the heart are indistinct, and a "whirring" murmur like that of varicose aneurism is sometimes heard when the cavities have been penetrated, as mentioned by Jobert; but, manifestly, this phenomenon belongs exclusively to cases where a communication exists between two of the chambers, as justly remarked by Mr. West. The percussion phenomena vary between complete dulness, where the pericardium is full of blood, and tympanitic resonance, where it is distended with air, whether this be obtained directly from the exterior, or indirectly by communication with the lungs or alimentary canal. In the latter case there will be cough and hæmoptysis, or hæmatemesis and passage of blood by stool. Copious hæmorrhage from the ex-

ternal wound does not prove that the heart has been implicated, as it may proceed from wound of the internal mammary or an intercostal artery, or from a wound of the lung. Bleeding from any of these sources, but especially from the heart itself, whether penetrated or not, is often profuse and quickly fatal. Oftener still it ceases on the occurrence of syncope, only to return with reaction. The external bleeding may be trivial, even whilst a fatal hæmorrhage into the pericardium is actually in progress. The mind is usually clear to the end. After the second day the danger of death from hæmorrhage, whether by syncope or by mechanical arrest of the heart's action from clot in the pericardium, may be regarded as past. From the second to the fourth day, however, pericarditis may be apprehended, and, when it does supervene, its presence will be announced by physical signs identical with those which characterize the idiopathic form of the affection, allowance being made for the possible presence of blood or air within the sac of the pericardium. The results are likewise similar; that is to say, adhesion, serous effusion, suppuration, or ulceration, may follow traumatic inflammation of the pericardium as they do that which arises idiopathically. The two latter results are, however, more common when a wound has been the cause of inflammation; because, in such case, air has found access to the serous cavity, and the irritation arising from its presence has been aggravated by that of decomposing blood.

Penetrating wounds, if not immediately fatal, may give rise to endocarditis, and, more remotely, to disorganization of the valves; to softening and ulceration of the heart, or to aneurism of its walls. Finally, embolism of the systemic or pulmonary arteries may likewise arise from this cause.

The prognosis is always unfavourable, whether the wound be penetrating or not; a fatal result may, and generally does, follow wounds of the parietes of the heart implicating a coronary artery, the opinion of Senac to the contrary notwithstanding. Where primary hæmorrhage is not fatal, the proximate or remote consequences usually lead to early dissolution.

Chassaignac mentions a case in which the apex of the heart was torn off by a pistol-ball. There was no external hæmorrhage

or hæmoptysis; but the patient died of pericarditis on the fifth day.*

Nevertheless, examples of complete recovery from wounds of the heart have been fully authenticated. Brugnoli, of Bologna, gives the case of a man who was stabbed with a knife in the left side of the chest, and died nineteen years and seven months subsequently. A scar was found on the anterior wall of the right ventricle, and the corresponding parts of the septum ventriculorum and mitral valve, so that the heart must have been all but transfixed.†

Mr. Callender has likewise recorded an example of recovery after a deep wound of the heart. A young man who had a needle fixed in his coat, had it driven into his chest in a scuffle. Nine days afterwards he applied at St. Bartholomew's Hospital, complaining of palpitation and pain extending from the apex of the heart to the left axilla, and along the inner side of the left arm as far as the elbow. Chloroform was administered, and an incision having been made in the fifth intercostal space, where a slight prominence was felt, a needle, nearly two inches long, was extracted. The point of the needle had manifestly been deeply fixed in the apex of the heart, for, after the "eye" had been exposed by division of the superficial structures, it described curves forwards and inwards, corresponding to the movements of the heart. Pain ceased immediately on the removal of the needle, a single drop of blood only oozed from the puncture, and perfect recovery followed at the end of four weeks.

Bullets may be encysted in the substance of the heart for many years. Bálch reports a case where a bullet was lodged in the heart for twenty years,‡ and Professor Hamilton, of New York, gives another, in which a bullet lay in the apex of the heart for thirty years.§

Latoni gives the following case. A soldier received a gun-shot wound in the chest, from the effects of which he completely recovered. Six years afterwards he died of a disease unconnected

* *Gazette des Hôpit.* September 4th, 1858.

† *Presse Méd. Belg.*, July 20th, 1862.

‡ *American Journal of the Medical Sciences*, July, 1861.

§ *Medical Times and Gazette*, January 26th, 1867.

with his former injury. At the examination of the body, a bullet was found encysted in the wall of the right ventricle, near the apex of the heart, and resting upon the septum ventriculorum.* Finally, in the hearts of animals of chase, and in game of various kinds, bullets have been found, which must have been lodged in the substance of the organ for a considerable period, without causing permanent impairment of the health or activity of the animal. Of this, the case of the stag mentioned by Harvey, in the heart of which a bullet was found lodged, furnishes an example.

Dupuytren insists upon the possibility of superficial wounds of the heart not giving rise to serious hæmorrhage, owing to the peculiar arrangement of the fibres by which such wounds are likely to be rendered valvular.† In contrast to such wounds, Mr. Adams‡ directs attention to those implicating the aorta or pulmonary artery, which are invariably and rapidly fatal, from the absence of any such arrangement of their fibres by which hæmorrhage from them could be arrested. In support of this statement, he quotes the case of a young woman who died from a penetrating wound of the aorta by a needle, one hour after the accident. Three or four punctures were found in the aorta, half an inch above the valves. The pericardium contained about a pint of clotted blood.§

In such cases as those above mentioned, in which foreign bodies are lodged in the substance of the heart, acute thoracic inflammation, even though apparently the most trivial, is likely to assume a very unfavourable character and prove rapidly fatal. Injury to the lung or pleura most seriously aggravates the danger of wounds of the heart.

In the *treatment* of wounds of the heart, exploratory probing should be scrupulously avoided, as likely to be mischievous, by dislodging clots, and favouring a return of hæmorrhage.

The positive indications are, firstly, to favour the formation of a clot in the wound, with the view of staying the further loss of blood; secondly, to prevent its dislodgment when formed; and,

* *Diction de Méd. et de Chirurg. Pratiques*, tom. iv., p. 258.

† *Leçons Orales*.

‡ *Dublin Medical Press*, April 3rd, 1861.

§ *Medical Circular*, January 25th, 1860; and *Dublin Hospital Gazette*, February 1st, 1860.

thirdly, to watch for, and promptly meet, symptoms of inflammation.

With the first mentioned object in view, ice should be applied over the wound, which must be quickly closed, and not afterwards disturbed, unless great dyspnoea occur, or other evidence of copious hæmorrhage into the pericardium be presented. In that case it may be necessary cautiously to remove the clots, which impede the motions of the heart. Foreign bodies should be carefully removed, without violence, and, as far as possible, without causing further loss of blood. Complete rest should be enjoined, and restorative measures *cautiously* adopted in view of the danger of secondary hæmorrhage from reaction. Acetate of lead should be given to favour coagulation, and morphia, by the mouth or sub-cutaneously, to moderate nervous excitement. Leeches may be necessary to avert or subdue subsequent inflammation, but general blood-letting should be deprecated. With the same object in view, mercury should be given. In the after treatment of the case, it may be necessary to remove serous effusion from the pericardium by mechanical means; but, this should be done with caution, considering the danger of secondary hæmorrhage from disturbance of the clot in cases still recent, and of exciting primary or secondary inflammation.

The weapons, or implements, with which wounds of the heart have been inflicted are of the most varied character. Thus, knives of various kinds, swords, bayonets, stilettoes, cobbler's awls, sharp iron bars, reaping-hooks, needles, thorns, fish-bones, and fractured ribs, have been the agents employed, or accidentally brought into requisition.

Mr. Adams gives the particulars of a case of wound of the heart by a fractured rib. The subject of the accident was the riding mare of a medical man. The animal, whilst moving at a rapid pace, fell under its rider, a heavy man, but was able to resume the journey. Twenty-five minutes afterwards it staggered and fell, and quickly died in convulsions. A double fracture of one of the left ribs was found, and the sharp end of the detached fragment had entered the pericardium, and wounded the coronary vein, whence a fatal hæmorrhage had proceeded.*

* *Loco citat.*

Fischer gives a table of 452 cases,* 87 of which he divides into three categories, according to the period of advent of syncope. Of these 87 cases, syncope was immediate in 30, and lasted from two minutes to four hours; in 38 instances, collapse was postponed for a short period; in 19 instances it was still later in appearing, and was due probably to dislodgment of the clot; it occurred in a few cases so late as the twenty-eighth day, when it was fatal.

In regard to seat of injury, Jamain, as quoted by West, gives the particulars of 121 cases.

OF THESE, THE

Right ventricle was wounded in	43 instances.
Left ventricle	28 "
Right auricle	8 "
Left auricle	2 "
Point and base of heart	7 "
Interventricular septum	2 "
Both ventricles	9 "
Both auricles	1 "
Left auricle and ventricle destroyed	1 "
Whole heart destroyed	1 "
Coronary artery	2 "
Without precise definition of seat	5 "
With no information as to site	12 "

121

Of Olivier's 61 cases, the right ventricle was the seat of injury in 23, and the left ventricle in 12 instances.

The following Table, intended to show, first, the seat of injury; secondly, the nature of the wound; thirdly, the period of subsequent survival of the patient; fourthly, the immediate cause of death; and, fifthly, the proportion of recoveries, has been constructed from the statistics of Mr. West, the records of the Dublin Pathological Society, and from *The Medico-Chirurgical Transactions of London*, conjointly.

* *Holmes' Surgery*, second edition, vol. ii.

TABLE IX.—WOUNDS OF THE HEART (TWENTY-NINE CASES).

Author.	Site of Injury.	Nature of Wound.	Period of Survival.	Cause of Death.	Recovery.
Mr. Adams .. <i>Arch. Générat</i> , 1843	Left ventricle	Penetrating, with round slug from fowling-piece	Half an hour	Hemorrhage into pericardium and pressure upon the heart	Recovery in 4 weeks.
Dr. Babington ..	Sigmoid flexure of colon, stomach, liver, diaphragm, heart, and right lung	Penetrat. "through and through"	19 days	Abscess of lung	Recovery.
Mr. Callender ..	Apex	Penetrating, from sewing needle	15 hours	Hemorrhage	
Mr. Le Gros Clark ..	Apex (left ventricle ?)	Lacerated by needle	Several hours	Hemorrhage	
Dupuytren ..	Left ventricle	Lacerated by broken rib	
Erlangen Museum ..	Heart	Non-penetrat. by frag. of sternum	1 year	..	
Dr. Ferguson ..	Left ventricle and septum	Penetrating, from esophagus by thorn of <i>Prunus Spinosa</i>	8 days	Hemorrhage into pericardium and pleura	
Dr. Fleming ..	Left coronary artery	Superficial, with cobbler's awl	10 minutes	Slow hemorrhage	
Dr. J. F. Fraser ..	Left ventricle	Penetrating and transfixing from penknife	34 hours	..	
Dr. J. H. Grant ..	Right ventricle, then diaphragm and intestines	Penetrating, gunshot	26 days	Asthenia	
Dr. Graves ..	Right ventricle, then diaphragm and stomach	Penetrating, from revolver-bullet	8 days	Abscess of heart	
Mr. J. Hamilton ..	Left ventricle	Penetrating and transfixing, from revolver-bullet	A few minutes	Hemorrhage into right pleura	
Mr. Hennen ..	Right a. and v., left a., and base of right lung	Pin wound	3 months	Carditis	
Hosp. Sick Children	Right ventricle	Needle, from right bronchus	8 days	"A fit"	
Hortius ..	Septum ventriculorum	Non-penetrating knife wound	2 hours	Hemorrhage from wound of coronary artery	
M. Lamotte ..	Both ventricles	Oblique non-penetrating, of both ventricles	63 hours	Ditto	
Baron Larrey ..	Both ventricles	Ditto	9 months	Pleurisy, pericarditis, pneumonia	
Dr. Leaning ..	Both ventricles, and septum	Penetrating, of needle	
Mr. Morton ..	Left base	Punctured with penknife	10 years	Anasarca and gangrene	
Dr. Mühlig ..	Both ventricles, and septum	Penetrating, stiletto	Some time	Shock	
Dr. Neill ..	Left ventricle	Non-penetrating, of needle	24 hours	Hemorrhage into pericardium and pleura, and between pleura and chest-wall	
M. Nelaon ..	Right ventricle	Contused, from pistol-shot	Immediate death	Pericarditis	
Prof. E. W. Smith ..	Right ventricle	Penetrating, but not transfixing, from clasp-knife	5 days	Slow hemorrhage	
Mr. Spencer Smith ..	Apex of right ventricle	Non-penetrating	4 days	Hemorrhage	
Mr. Thompson ..	Left ventricle	Penetrating	4 years & 5 months	Phthisis pulmonalis	
University College Hospital	Descending coronary vein	From fish-bone, passing out of esophagus, and through diaphragm and pericardium	Immediate death	Hemorrhage	
Mr. West ..	Anterior wall of right ventricle	Inclined, 14 lines deep, and non-penetrating	25 days	Suppuration, and hectic	
" ..	Both ventricles	Extensive, lacerated, by end of broken ribs and sternum	
Mr. Wood ..	Right auricle and ventricle	Lacerated and penetrating	

The subject of *malformations of the heart* would be more appropriately discussed in a special treatise on pathology, than in a work designed mainly to present a clinical exposition of cardiac disease. However, it demands some notice, both because it is the necessary complement of any work on this subject designed to be even approximately complete, and because it represents a disturbing element in diagnosis.

Andral and Paget have classified congenital malformations of the heart under the three heads of "defective," "excessive," and "perverted" development.

Peacock, whose work* constitutes the great repertory of our knowledge of this subject, following Breschet, adopts the division into "misplacements," "malformations," and "irregularities," both in regard to the heart and the great vessels connected with it.

Misplacements are of two kinds; viz., (a) within the thorax; and (b) outside the thorax.

Malformations he regards as essentially due to arrest of development, and he classifies them according to the period of foetal life at which the arrest occurs, into: (a) Those due to arrest of development from the fourth to the sixth week; scarcely any trace of the septum between the right and left chambers as yet existing, the aorta and pulmonary artery imperfectly evolved, or the primitive arterial trunk even still retained. (b) Those which arise from arrest of development occurring from the sixth to the twelfth week; the septa being imperfectly formed, and the aorta and pulmonary artery more or less perfectly developed. (c) Those which are due to arrest at a still later period; the septa being complete, and the origin of the vessels normal, but in which from disease, the ordinary *post partum* changes have not taken place.

"Irregularities" are of two kinds; viz., (a) those arising from premature closure of the foetal passages, and (b) from anomaly in the number or form of the valves.

Imperfection of the pericardium has been illustrated by several cases recorded by Bristowe, Baly, Peacock, and others. In these the pericardium was represented by a crescentic fold, placed usually behind the heart; it is the product of arrested develop-

* *Malformations of the Human Heart*, second edition, 1866.

ment of the pericardium from the sheath of the great vessels, in the direction downwards.

A *biloculate* condition of the heart has been repeatedly met with. In some cases rudimentary septa existed, and one or two ventricular vessels were always found. When only one, it corresponded to the aorta or pulmonary artery, according to the ventricle represented by the vessel actually present; and from this single vessel systemic or pulmonic branches were given off, as the case might be.

In *triloculate* hearts there are two auricles and one ventricle, the auricular septum being imperfect, and that of the ventricles either entirely absent or rudimentary. There are, in such cases, usually two auriculo-ventricular openings, but sometimes only one. Most frequently two vessels arise from the common ventricle; but, occasionally one only is so given off, representing the aorta, and from this the pulmonic vessels are supplied with blood through the ductus arteriosus. Patients with this anomaly have lived to the age of twenty years.

In the *quadriloculate* heart, the auricular, or the ventricular septum, or both, may be imperfect. The defect is most frequently found in the "undefended space" at the base of the interventricular septum, where naturally the wall consists only of two layers of endocardium, some fibrous tissue on the left side, and a few muscular fibres on the right. In this situation the opening is usually triangular, but occasionally it is oval or round. The aorta and the pulmonary artery are distinct, but the latter is often rudimentary, the pulmonary vessels being derived from the ductus arteriosus. The foramen ovale, as well as the ductus arteriosus, may be pervious; the ventricular septum is misplaced, usually deviating to the left, so that the aorta arises in part or wholly from the right ventricle, and the latter chamber, having imposed upon it the duty of carrying on the double circulation, is greatly thickened and dilated, whilst the left ventricle is imperfectly developed or rudimentary.

Hunter was of opinion, and Peacock likewise holds, that the primitive defect consists in contraction of the pulmonary artery, in consequence of which the septum, still incomplete, is pushed to the left by the great pressure of the blood, whilst the aorta is

drawn to the right, the passage of blood preventing the completion of the septum.

Meckel, on the contrary, maintains that defect and deviation of the septum are the primary anomalies; the blood of the right ventricle, finding a ready escape through the aorta, the pulmonary artery remains undeveloped.

The obstruction may be located at the infundibulum, and consist of thickening of the endocardium and hypertrophy of the muscular wall; it may exist at the arterial ring, from contraction of the fibrous zone; at the free edges of the valves by cohesion of the segments, or in the artery beyond the valves.

An example of perforate interventricular septum, unaccompanied by any other lesion of the heart, has been published by Dr. Habershon.* The subject was a man, aged fifty-four, of intemperate habits. He was attacked with cough, dyspnoea, and hæmoptysis, after exposure to cold; dropsy quickly followed. A systolic murmur was heard to the right of the sternum, at the ensiform cartilage, and in the left axilla and back. After the man's death, a funnel-shaped opening, a quarter of an inch in diameter, was found in the base of the septum ventriculorum. That a current passed through this opening, from the left to the right side, was concluded both from the conformation of the passage, and from certain markings exhibited by a fibrinous deposit upon the adjacent segment of the tricuspid valves. In reference to cases of this kind, Dr. Habershon remarks, that the patients may enjoy tolerably good health till an attack of bronchitis deranges the pulmonary circulation.

Atresia of the pulmonary artery may be due to the presence of a septum or diaphragm at the orifice, or to atrophy and closure of the vessel itself. In such cases, the ventricular septum may be incomplete; the aorta communicating with the right ventricle, and carrying on the pulmonary circulation through an open ductus arteriosus, the right ventricle being hypertrophied, and the left auricle and ventricle atrophied. These cases are the most numerous. In other instances the ventricular septum is complete, whilst the foramen ovale is open, and through it and the ductus arteriosus the pulmonary circulation is carried on;

* *Guy's Hospital Reports*, vol. xvii., 1872.

but when, in these cases, the ductus arteriosus is impervious, the circulation is maintained through supplementary branches from the aorta or left subclavian artery supplied directly to the lungs, or through dilated bronchial arteries. In these latter cases the right ventricle is only rudimentary, and closure of the pulmonary artery took place, presumably, after completion of the septum ventriculorum.

The subjects of these malformations rarely survive even for a few days; but, in one such instance at least, life has been prolonged to the tenth month.

Partial or complete closure of the right and left auriculo-ventricular orifices, and of that of the aorta, but not in the same subject, have been repeatedly demonstrated. Where the tricuspid orifice was partially or totally obstructed, the circulation was carried on through an open foramen ovale and ductus arteriosus, the latter receiving its supply through the aorta. In cases of obliteration of the mitral opening, the passage of blood was from the left to the right auricle; the foramen ovale and ductus arteriosus being patulous, and the latter receiving its blood from the aorta by inversion of the current, as in the former case, whilst the aorta arose from the right ventricle.

Where the aortic opening was closed, the circulation had been carried on from the left to the right ventricle through an imperfect ventricular septum, and thence through a patent ductus arteriosus into the descending aorta; here the current was divided into two portions, one conducted upwards through the arch and into its branches, and the other downwards. The subjects of these malformations usually die within a few days of birth.

Premature closure of the foramen ovale and of the ductus arteriosus is not rare, but both lesions are seldom witnessed in the same subject.

In the former case, the right auriculo-ventricular opening, and that of the pulmonary artery, are very large, the right auricle is dilated, and the right ventricle dilated and hypertrophied; the left auricle and ventricle are hypertrophied, the cavity of the latter nearly obliterated, and the left auriculo-ventricular opening proportionately reduced in diameter. The systemic circulation, which is highly venous, is carried on mainly through the

ductus arteriosus. Infants so affected have lived only a few hours.

When the ductus arteriosus has been prematurely closed, the foramen ovale has remained open, and the ventricular septum imperfect. In such cases the right ventricle freely communicates with the aorta; both are very large, as likewise are the tricuspid orifice and the right auricle, whilst the left cavities are much reduced. A minute dribble of blood is supplied to each lung through the corresponding lateral branch of the pulmonary artery. Persons so affected rarely live beyond a few days. In some instances life has been prolonged to several months, and in one case to sixteen years.

Patency of the foramen ovale may arise from: (a) disproportion between the opening and the valve; this may be the consequence of absolute enlargement of the opening, of imperfect development or absence of the valve, or of both combined; (b) from perforation of a valve otherwise competent; (c) from laxity of a well formed and fully proportioned valve, owing to excessive length of the cornua; and (d) from simple non-adhesion of the valve.

Examples of the first three kinds are rare, and are associated with other serious malformations of the heart, whilst instances of the last are sufficiently common.

Doctor Mayne has recorded an interesting case of imperfection of the inter-auricular septum, the result of congenital deficiency of the operculum at the foramen ovale. A female, aged thirty-five years, suffered from cardiac distress, accompanied by obscure cardiac symptoms. There was wandering pain in the chest, accompanied with palpitation and short dry cough, and the countenance was pale (not cyanotic). Respiration was tranquil. The pulse was regular, soft, small, and ranging from 80 to 90 in the minute. The patient was lethargic, and slight exertion quickened the pulse and the respiration. The jugular veins were not distended, nor did they pulsate, and there was no dropsy. Perspiration was constant and offensive, but other excretions were scanty; appetite indifferent. General pulmonary resonance; respiratory murmur feeble, and but slightly augmented by deep inspiration or by coughing; no râles; precordial dulness extended; cardiac rhythm normal, and no fremitus. A

loud *bruit de soufflet*, accompanying the first sound, was localized at the sternal end of the left fourth costal cartilage. The second sound was distinct and normal. The patient lived two years subsequently, and died, suddenly, whilst crossing the court yard of the hospital.

The right auricle was found to be thick, and of twice its normal capacity; an opening as large as a half-crown piece existed in the septum auricularum. The edges of this opening were smooth, and not even the rudiment of a valve existed in connexion with it. The venæ cavæ were small, the tricuspid orifice was large, and the right ventricle, which alone formed the apex of the heart, was dilated and thickened. The pulmonary artery and its orifice were very large. The left auricle was of the usual size, but the pulmonary veins were large. The left ventricle was small, and its walls were thin. The mitral orifice was contracted, and the mitral valve thickened. The aortic orifice was small, and the valves healthy. The ductus arteriosus was pervious.* In this case contraction of the mitral orifice was evidently the primary malformation, and the blood must have circulated from the left to the right side of the heart, through the open foramen ovale, whilst a very minute quantity only passed through the left ventricle.†

In reference to the foregoing most interesting case, Dr. Mayne remarks, that the feeble character of the respiratory murmur was most probably to be attributed to the arterial quality of the blood circulating through the lungs. In this opinion I quite concur. From the rhythm and seat of the murmur heard, and the small quantity of blood that must have passed through the mitral orifice at each systole of the left auricle, I conclude that the murmur was due to the passage of a current from the left into the right auricle, through the open foramen ovale. In that case, however, I would have expected that its rhythm should have been presystolic.

Peacock holds that closure of the foramen ovale is due partly to contraction of the opening, and partly to shortening and approximation of the cornua, by the oblique and horizontal bands

* *Dublin Journal of Medical Science*, vol. i., 1841, and *Proceedings of the Dublin Pathological Society*, vol. ii., p. 35.

† See J. F. Meckel, *Tabulæ Anatomico-Pathologicae*, fig. iii., plate i.

of muscular fibre described by Senac as intersecting the cornua, and running across the valve parallel to its free margin. The contraction of these fibres results partly from distension of the left auricle after birth, and in some degree also from stimulation by contact of arterial blood.

Persistence of the ductus arteriosus may result from: (a) obstruction in the lungs at the time of birth; the foramen ovale will, in this case, be likewise open; (b) obstruction at the left auriculo-ventricular opening; the foramen ovale is also open in this case;* and (c) obstruction at the orifice of the aorta, or in the descending portion of that vessel, the ventricular septum being also defective.

Excess or deficiency in the number of the semilunar valves may arise from congenital malformation, or from subsequent disease. The former is less frequent, and, when present, it is usually found at the pulmonic orifice. Of 41 such cases reported by Peacock to the Pathological Society of London in 1851, 9 only were examples of excess in the number of segments, and 8 of these were located at the orifice of the pulmonary artery.

Excess in the number of segments seldom gives rise to obstruction, and, scarcely ever, to incompetency of the valves. Excess is, therefore, less often associated with serious organic disease of the heart than is defect in the number of segments, the latter being very seldom met with unaccompanied by the general results of valvular obstruction or incompetency.

The valves, both auriculo-ventricular and arterial, may be represented by a single curtain with a round or triangular aperture; or by two segments, one of which is duplicate by fusion. Disease may, however, give rise to corresponding changes.

Atrophy of the valves, by defect of the fibrous layer, or of the serous investment at the free edges, may also be met with.

Irregularity in the origin, course, and distribution of the aorta and pulmonary artery is rare. The vessels have been found transposed, the aorta arising from the right, and the pulmonary artery from the left ventricle. This condition is usually associated with patency of the foramen ovale and ductus arteriosus, and with an imperfect state of the septum ventriculorum. It may or may

* *Vide* Dr. Mayne's case, *antea*.

not be accompanied by complete transposition of the viscera; more frequently it is not so accompanied.

The most common anomaly of these vessels consists in partial or complete closure of the aorta at the point of junction of the ductus arteriosus; the lower portion of the body being supplied with blood, wholly or in part, through the latter vessel.

Defect or redundancy of the venæ cavæ, and of the coronary vessels, and irregularities in their course and connexions, are rare.

Cyanosis, cyanopathia, morbus cæruleus, or the *blue disease*, has been attributed by pathologists to two different, though frequently associated, causes; namely, general venous congestion, and intermixture of the venous with the arterial blood.

The former doctrine was propounded by Morgagni, and supported by Corvisart, Louis, Laennec, Cruveillier, Valleix, Hasse, Rokitsky, Joy, and Moreton Stillé. The latter, by Dr. Hunter, Meckel, Gintrac, Paget, Williams, Hope, J. Crampton, and Walshe; also, with some modification, by Bouillaud and Forget. Peacock adopts the former view, which is also mine.

Peacock attributes the greater degree in which cyanosis is manifested in cases of malformation, to its being in such cases congenital; the integument being thinner, and the capillaries more dilatable in the infant than at a later period of life.

Cyanosis is most frequently congenital, or exhibited in the first years of life. In the latter case it is either developed gradually, or manifested quite suddenly. In mature age, however, it may be primary; it is then the result of shock, or of a severe blow upon the chest. According to Stillé* it has been met with in association with the following lesions and anomalies.

1. Dilatation and hypertrophy of the right chambers of the heart.
2. Contracted state of the left chambers.
3. Heart with only one cavity.
4. Heart with two cavities, an auricle and a ventricle.
5. Heart with three cavities, two auricles, and one ventricle.
6. Persistence of the foramen ovale, or a cribriform auricular septum.
7. Perforation or absence of the ventricular septum.

* *American Journal of the Medical Sciences*, new series, vol. viii., 1844.

8. Dilatation of the aorta.
9. Obstruction at the orifice of the pulmonary artery from contraction, or from adhesion or deficiency of the valves ; from cartilaginous or other growths at the orifice ; or from closure by membranous septa.
10. Persistence of the ductus arteriosus.
11. Contraction of right auriculo-ventricular opening.
12. Transposition of the aorta and pulmonary artery.
13. Origin of these vessels from one ventricle.
14. Origin of these vessels by a common trunk.
15. Aorta supplying heart and upper limbs, and pulmonary artery supplying remainder of body.
16. Aorta giving off the pulmonary branches.

He has examined upwards of 80 cases, and adverts to 5 out of this number, in which cyanosis existed, without any lesion or defect which would admit of intermixture of the venous and arterial blood. He arrives at the following general conclusions.

1. There is no proportion between cyanosis and the degree in which the blood is mixed.
2. Complete admixture of the blood may take place without cyanosis.
3. Cyanosis depends essentially upon any cause which, acting at the centre of the circulation, is competent to produce capillary venous congestion.

The most frequent cause is obstruction of the pulmonary artery, which, in some form, existed in all the cases collected by him, except 9 ; in these, an equivalent condition was found in the existence of other organic lesions.

Out of a total of 40 cases of congenital cyanosis, the pulmonary artery was partially obstructed or impervious in 28.

In reference to the duration of life in the congenital cases, Stillé remarks that death took place before the age of twenty years in 27 instances out of 40.

I am decidedly of opinion that cyanosis depends essentially upon capillary venous congestion, to whatever cause this may be due ; and in holding this opinion I am happy to find myself in accord with two such eminent writers as Drs. Stillé and Peacock.

It may be due to causes apparently the most opposite, but all

leading directly or indirectly to systemic venous congestion. I have known it to be caused by patency of the foramen ovale; serous effusion into the pericardium and both pleuræ; enlargement of the thyroid gland, both benign and malignant; and aneurism of the ascending aorta opening into the descending cava.

The case just alluded to (see "Aneurism," case of Michael W.), presented the most remarkable example of cyanosis which I have witnessed. The upper part of the body of the patient, a young man, was literally black, as if it had been thickly smeared over with dilute ink.

Even in cases of congenital malformation of the heart, leading inevitably to admixture of the venous with the arterial blood, it will be found, that although the ordinary condition of the body is one representing an imperfect circulation in respect to temperature, ability to bear cold, tint of cutaneous and mucous surface, and activity of secretion and of mental faculties, yet that cyanosis, or positive blue discoloration of the surface, is presented only when the mind or body is under excitement, *i. e.*, when general venous congestion is produced by quickened circulation.

Irrespectively of cyanosis, which is a symptom of much too general import to possess any special diagnostic value, congenital malformation of the heart, involving direct communication between its opposite sides, is characterized by certain general features which can scarcely be misunderstood. These are indicative of a circulation, not only feeble and imperfect, but liable to paroxysmal derangement of an aggravated character. Thus, the temperature is below the normal standard, secretion is sluggishly performed, the mental faculties are dull, the surface is pallid or dusky, and the temper is either singularly passive, or morbidly irritable. Under excitement of any kind which is capable of quickening the circulation, the surface of the body, and especially that of the face and neck, becomes swollen and livid, the breathing rapid and laboured, and the action of the heart tumultuous; convulsive seizures, not unfrequently fatal, are of ordinary occurrence, under such circumstances.

Sir Dominic Corrigan exhibited before the Dublin Pathological Society* the heart of a girl, aged eleven years, who had been cyanosed at intervals from her birth. The foramen ovale was

* January 9th, 1858.

patent. During the patient's last illness it was remarked that cyanosis appeared only when she sat up ; in the recumbent posture the colour of the surface was natural. This, he remarks, together with the absence of capillary congestion, distinguishes true cyanosis from that due to simple congestion.

A murmur may, under these circumstances, be likewise developed, the rhythm and site of which will depend upon the lesion actually present. I do not think a murmur is necessarily associated with patency of the foramen ovale. I have not met with a single example of it which was clearly traceable to this anomaly as its cause. On the contrary, the three subjoined cases, in each of which, as judged by the symptoms and the *post mortem* appearances, a direct and free passage of blood from one auricle to the other must have taken place whilst the patient was under observation, afford negative evidence of the strongest kind.

Doctor J. W. Ogle furnishes some valuable evidence on this point.* He gives a list of 62 hearts examined with reference to the state of the foramen ovale. In 13 of these the passage was imperfectly closed ; it varied in size from a fissure or chink due to non-adhesion of the valve, to an oval or round aperture admitting the tip of the little finger. In none of these cases was there evidence of ulceration, or appearance of rupture of the valve, to account for the opening. The ages of the patients varied from sixteen to sixty years, the greater number being between thirty and forty years. Murmur was not noted in connexion with any of these cases, except one ; in that solitary instance the murmur was diastolic, loudest at the root of the aorta, and manifestly due to inadequacy of the aortic valves, which was proved by *post mortem* examination to have existed. In 6 of these 13 cases, in all of which death was caused by various diseases unconnected in several instances with the organs of circulation, the heart had been carefully examined during life, and it is positively averred that no murmur whatever existed.

A seventh example of similar import is quoted from Dr. Ogier Ward. It was that of an infant which died from symptoms of suffocation eleven days after birth, having been previously cya-

* *British Medical Journal*, June 13th, 1857.

notic; on this account the heart and lungs were carefully auscultated, but no murmur was detected. A congenital communication between the trachea and œsophagus was found after death. The pulmonary artery was dilated, and the ductus arteriosus and foramen ovale were patent.

As regards the *treatment* of cases of malformation, little needs to be said. It may be briefly summed up in the general rule, to avoid all causes of excitement, maintain a moderate temperature, promote the secretions, administer nutritious and digestible food, and, where congestion is extreme and threatens life by its excess, to draw blood in moderate quantity, by means of one or two leeches. Due precaution should be observed against dangerous or fatal hæmorrhage, owing to the debility of the patient, and the occasional difficulty of arresting the bleeding.

Subjoined are the details of three examples of this condition.

CASE LXXI.—Cyanosis; Partial Suppression of Arterial Pulse in the Right Side of Head and Neck, and Right Upper Extremity; General Dropsy; Death. Fatty Degeneration of the Heart; Concentric Hypertrophy of the Left Ventricle; Patency of the Foramen Ovale.

Kate B., aged twenty-five years, was received into hospital, February 24th, 1865. For some years previously her breathing had been short, and six weeks prior to admittance she caught cold and had cough with expectoration; after a few days her breathing was greatly oppressed, and her feet began to swell. For three months she had not menstruated. Decubitus was exclusively sinister. The face was bloated, and of a purple colour, feet and legs swollen, right hand and forearm also cedematous, ascites, suffocative cough with mucous expectoration. Expiration prolonged. Pulse 132. The impulse of the heart was rather vigorous, but regular, and its sounds were normal. The supra-clavicular fossæ were obliterated on both sides, and on the right side especially the jugular veins were greatly distended, but without pulsation; the apex of the lung was very prominent above the clavicle, and the superficial veins of the shoulder and chest were distended on the same side. On the left side of the

neck there was venous pulsation. The chest was universally resonant, and crepitant râles were everywhere audible. A blister was applied to the chest, and small doses of ammonia and spirit of chloroform were given.

On the 26th, the observation was made that carotid pulsation could not be felt on the right side, whilst it was distinctly perceptible on the left. Œdema had disappeared from the right hand, but continued in the forearm. The bases of both lungs were crepitant, but not dull on percussion. Over the right clavicle and sub-clavicular space there was a shade of dulness. There was no difference between the right and left radial pulse, or in the pupils, and no impairment of voice.

March 6th. Pulse in left arm 120 and weak; scarcely to be felt on the right. Face greatly congested, especially on the right side, and carotid pulsation scarcely detectable by the finger or the ear on that side, whilst it was strong and visible on the left. Over the left apex of the heart there was a faint systolic murmur, but this was not elsewhere audible. Patient generally weaker, cough more troublesome. Urine free from albumen. The following mixture was now given: *R* Sulph. quiniæ, gr. xij; Sulph. morphinæ, gr. j; Acid. sulphur. dil., ℥x; Aquæ font. q. s. ad 3vj. A tablespoonful to be taken every third hour, together with four ounces of wine within the same period. It was manifest that pressure was being made upon the arteria and right vena innominata by some solid body.

7th. Arteries on left side of neck bounded with great violence, but no pulsation existed on the right side. Death took place in the course of that day.

The body was examined ten hours after death. The thoracic viscera were alone inspected. There were some old adhesions of the right lung, and in the apex of the left, which was puckered on the surface, a small mass of cretified tubercle was found. There were about two pints of serum in the left pleural cavity, and about half that quantity in the right. The bronchial membrane was congested. A mass of enlarged lymphatic glands occupied the upper portion of the anterior mediastinum, and was found to press upon the arteria and right vena innominata, and upon the descending cava. This circumstance afforded the only rational explanation

of the partial congestion and cedema of the right side, and of the partial suppression of pulse in the right carotid and subclavian arteries. The heart was somewhat enlarged in its transverse diameter, and its apex was blunted by excessive growth of the left ventricle. The right cavities were normal as to capacity and thickness of walls. The left ventricle was greatly hypertrophied, its wall, a little below the base, being one inch thick, and its cavity contracted. The substance of the heart was pale in section, and some muscular fibres from the left ventricle, examined microscopically, were found in the medium stage of fatty degeneration. The fibres retained their normal outline, but were composed almost entirely of fat molecules. In a few situations, transverse striæ were faintly visible. The valves were all healthy and competent. The interior of the aorta, throughout the arch, presented white streaks, as in the early stage of atheromatous change. By an oversight on the part of my resident pupil, the roots of the great cervical vessels, with the adjacent glands, were not preserved for examination. The foramen ovale was widely patent; the valve existed, but it was shrunken, and incompetent to close the opening.

On the day preceding death, and on that day only, a faint murmur of systolic rhythm was heard over the left apex. No murmur was elsewhere, or at any other time heard, although the patient had been daily and carefully examined. This murmur was certainly mitral in origin, and, according to my view as previously explained (p. 219), dependent upon mitral inadequacy produced by yielding of the walls of a softened left ventricle at the acme of systole.

The case may, therefore, be classed with those of Dr. Ogle, as evidence against the doctrine of systolic murmur from patency of the foramen ovale, even when this is accompanied by actual passage of blood directly from auricle to auricle. Direct transit of blood must have taken place in this case, at least during the few last weeks of life. Of similar import is the following case :

CASE LXXII.—*Hyperæmia of the Face and Neck; Orthopnœa and Hæmoptysis; General Dropsy; Albuminuria; Hyaline and Fatty Renal Tube Casts; Weak Action and Obscure Sounds of the Heart, but no Murmur; Death. Hypertrophy with Dilatation, and Fatty Degeneration of the Left Side of the Heart; Widely Patent Foramen Ovale; no Obstruction on the Right Side, and no Valvular Disease; Congestion of the Liver and Kidneys.*

Mr. L., aged forty-nine years, of strong frame, and apparently of robust constitution, was admitted into hospital as a private patient, paying a stipulated sum for his support and attendance, on the 14th September, 1865.* He had lived freely, but had not drunk to excess; he had not had gout or rheumatism, neither of which was hereditary in his family. For several years previously, he had experienced shortness of breath after active exercise, and about six months prior to admission he complained, for the first time, of palpitation. The breathing now became much oppressed, the lower extremities swollen, and he spat up a little florid blood on two or three occasions. He passed some weeks at Harrogate, where, according to his own account, a physician whom he consulted informed him that he was the subject of functional derangement, but not of organic disease of the heart.

When he first came under my notice, the following was his condition. Much nervous excitement; face rather florid, but not puffed; tongue clean; bowels constipated and distended with flatus; and some liquid in the sac of the peritoneum. Considerable oedema of the trunk, scrotum, and lower limbs. Urine passed in moderate quantity, and loaded with lithates; its sp. gr. was 1·020, and it contained some albumen. Pulse 120, not strong, but regular, and equal at both wrists. Orthopnœa, but no cough. Precordial dulness extended, especially upwards. No cardiac impulse to be felt; sounds rather dull and masked, but no murmur to be heard. The base of the right lung was dull, and

* The arrangement under which persons in affluence were received into the Mater Misericordiæ Hospital as "private patients" was found to be attended with great inconvenience, and was much abused by those for whose benefit it was intended; I rejoice to add, it has been long since abolished.

here respiration was associated with crepitus, whilst elsewhere percussion and respiratory sounds were normal, the latter somewhat exaggerated on the left side. There was marked bulging of the right supra-clavicular fossa, to which the patient referred as the seat of occasional pain and "beating," the former extending down the right side and arm. The swelling was, however, resonant on percussion, yielded loud respiratory sounds, and was manifestly caused by ascent into the neck of the apex of the corresponding lung. Pupils slightly contracted and equal. The case was deemed very obscure, and no positive diagnosis was made. A diuretic and aperient pill, consisting of blue pill, squill, and digitalis, was given thrice daily. The lower and back part of the right side of the chest was dry cupped, the use of liquids was restricted, and perfect rest was enjoined.

On the 18th he was much excited, and I discovered that he had secreted whiskey in his lock up, and taken about a pint of it in the course of the preceding night. Had spat up some florid blood. Prepuce punctured, and discharged a large quantity of serum. To have half an ounce of the following every third hour. \mathcal{R} Tinct. colchici, \mathcal{M}_{cxx} ; Potass. acetat., grs. cxx ; Infusi calumbæ, q.s. ad $\mathfrak{3}\text{vii}$. \mathcal{M} .

19th. Medicine not taken; fully a pint of sherry consumed since yesterday, despite the remonstrance of his attendants. Colchicum was now prescribed in form of pill, in combination with blue pill and compound squill pill.

20th. Had passed a sleepless night, owing to pain and spasm in lower part of right side, extending to the liver. Urine passed in moderate quantity; it was amber-coloured, acid, sp. gr. 1.020, and contained a good deal of mucus; it exhibited under the microscope octohedral crystals of oxalate of lime, a few epithelial cells studded with fat granules, some hyaline tube casts set sparsely with oil dots, and one of small size with perfect epithelium. Is much quieter, and feels better. Fæces devoid of biliary pigment.

25th. Spasm in region of heart last night. Urine increased in quantity to three pints, alkaline, and of sp. gr. 1.015. To have ox gall, blue pill, and co. squill pill in combination; afterwards a Seidlitz powder, and to be dry cupped over the heart.

Within the succeeding few days copious effusion of serum into the pericardium took place, causing great increase in the area of precordial dulness, which extended, on the 2nd October, up to the second intercostal space. Under the operation of a blister, and still more in consequence of an abundant discharge of serum through several small openings, formed by sloughing of the integument of the legs, this was much reduced on the 4th, and at that date likewise oedema of the genitals had entirely disappeared. The urine was of sp. gr. 1·020; it was usually acid, but occasionally neutral, and once, at least, decidedly alkaline; sometimes clear, frequently loaded with lithates, and in quantity about two pints in the twenty-four hours. Bowels generally constipated, and dejecta light in colour. During the last few days of his life respiration was greatly embarrassed, whilst the supraclavicular prominence, previously noticed on the right side had subsided. Sounds of the heart rather obscure.

Death took place on the 5th October, consciousness having been retained to the end.

The body was examined ten hours after death. *Abdomen.* Slight congestion of kidneys and of liver, the latter organ being somewhat enlarged. The kidneys, I regret to say, were not microscopically examined. *Thorax.* Right lung engorged, especially at the base, and dispersed throughout it were several nodules of extravasated blood. The heart was in excess of the average size; some fat on the surface. The foramen ovale was patent, admitting the point of the index finger. The left auricle and ventricle were hypertrophied. The right and left auriculo-ventricular orifices were much dilated. The pulmonary artery was somewhat in excess of the usual diameter, but of normal size relatively to the ventricle. The aorta was of average size. Ductus arteriosus not examined. The valves were all healthy. The muscular structure of the left ventricle exhibited under the microscope complete fatty degeneration of the fibres, the outlines being distinct, whilst of the greater number the centres were uniformly clear and structureless, presenting a few transparent oil dots. In many fibres the latter were disposed in linear order, some within, and others external to the sarcolemma. No striæ whatever were visible.

There must have been a free passage of blood through the open foramen ovale, as there was no valve; the current had most probably set in from the left to the right auricle, since the tint of the face was decidedly arterial, and the left auricle was hypertrophied. There was primary disease of the kidneys, and hypertrophy, with consecutive fatty degeneration and dilatation, of the left side of the heart.

The following case affords a further example of the same kind; viz., of passage of blood through the foramen ovale, without murmur.

CASE LXXIII.—*Rapid Pulse and Embarrassed Respiration; Tumultuous Action of Heart; Crepitant Râles on Right Side; Death. Tuberculosis of both Lungs, and Right Tubercular Pneumonia; Patency of Foramen Ovale from Non-Adhesion of Valve; Hypertrophy of Left Ventricle; Valves all Healthy.*

Eliza R., aged eight years, an inmate of St. Clare's Orphanage, Harold's Cross, was admitted into hospital under my care, May 9th, 1866. Had been delicate from birth, and a fortnight previous to admittance, was attacked with her present illness. She moaned constantly, the skin was hot and dry, and the lips were livid. Pulse 158. Respiration 60. The action of the heart was tumultuous; there was much tenderness in the precordial region, but no attrition-sound or cardiac murmur existed. Over the right back generally there was crepitant râle, but no percussion-dulness, and very slight cough. The right side was dry cupped posteriorly, and then poulticed with linseed meal and mustard, and small doses of grey powder and antimonial powder were given every third hour.

Owing to the advent of diarrhoea on the 10th, mercurial inunction was substituted for the preceding. The symptoms were then much ameliorated.

On the 12th, however, respiration was again greatly embarrassed. The face was remarkably congested, and of a leaden hue. Pulse 168. Right back still crepitant, but clear on percussion. A blister was applied here, and other treatment was continued.

The diagnosis of central pneumonia on the right side, and *probable* patency of the foramen ovale was now made; the condition of the patient was most unpromising.

She died on the afternoon of that day; and twenty-four hours subsequently the body was examined. The lungs were of large volume, gorged with blood, and studded throughout with minute tubercle, which, in the right lung, had become aggregated in a few places, and was in process of softening. The superior and the central portions of the right lung had become solid; they sank in water, but not completely, as they floated in contact with the bottom of the vessel. The remainder of the right, and the entire of the left lung, though engorged, tuberculosed, and retaining the volume of full inspiration, were resonant on percussion, and floated in water. There was some liquid in the pericardium. A large mass of decolorized fibrin occupied the right auricle, and extended into the right ventricle. The foramen ovale was patent. The spoon-shaped extremity of a large director readily passed through it from the right to the left auricle. The passage, which was covered by a valve, well-formed but non-adherent at its upper margin, led obliquely from below and from the right side upwards and to the left. The left ventricle was somewhat hypertrophied. The valves, both arterial and auriculo-ventricular, were all in a healthy condition. The spleen was rough upon the convex surface, and partially adherent to the diaphragm, on which, to a corresponding extent, minute, pale, granular deposits of lymph were observable. The liver was large but healthy, as were likewise the kidneys.

This case was an example of patency of the foramen ovale, entailing upon the child constitutional delicacy, but not giving rise to serious consequences, save, indirectly, by favouring tuberculization of the lungs, till the occurrence of acute tubercular pneumonia. The resistance then offered to the pulmonic circulation, by causing increased blood pressure in the right chambers, determined displacement of the operculum, and so caused passage of blood directly from the right to the left auricle. Cyanosis then, for the first time, appeared. The pulmonary complication was the immediate cause of death.

CHAPTER VII.

DISEASE OF THE LINING MEMBRANE OF THE HEART.

UNDER this head we will consider: (*a*) Endocarditis, and (*b*) Structural Disease of the Valves.

Endocarditis may be either acute or chronic. In the acute form it is met with almost exclusively in connexion with articular rheumatism, of which it constitutes the most formidable complication. It has occurred in 16 per cent. of Dr. Peacock's cases, or in the proportion of 1 : 5·9.* Bamberger estimates the proportion at 1 : 20.

It occasionally, though very rarely, constitutes the *only* manifestation of the rheumatic diathesis. I have met with a few such examples in persons decidedly rheumatic, as evinced by previous attacks of the disease in the ordinary form. It has been assumed also that acute endocarditis may occur in the simple idiopathic form, that is, unassociated with other antecedent or concurrent cardiac lesion, and not attributable to any of the usual constitutional causes. I have never met with an example which could be fairly adduced in support of this doctrine, and I question its truth.

It is not to be doubted that, in certain cases, pericarditis may arise from exposure of the body to cold, when over heated. Of this, Dr. Stokes has given a notable example, already referred to (p. 752). It occurred in the person of a young gentleman who, when much heated from athletic exercise, imprudently lay down and slept upon the ground without having dressed. But of endocarditis from this or any similar cause, there is not, as far as I know, an authenticated example on record.

In rheumatism, the liability to endocarditis is in direct proportion to the severity of the articular affection, and the number of joints engaged; *i. e.*, it is proportionate, presumably, to the

* *Medical Times and Gazette*, August 2nd, 1873.

quantity of the special morbid matter actually present in the blood. Next, in point of frequency, it occurs, although not so often as pericarditis, in connexion with Bright's disease, and in this association it is usually fatal. It occurs, likewise, very frequently in measles, and less often in scarlatina, small pox, puerperal fever, phlebitis, and pyæmia. In the two last mentioned connexions, the right side of the heart alone is usually engaged.

M. Lancereaux has described a special form of ulcerative endocarditis, which is produced, as he believes, by paludal miasm.* It usually engages the aortic valves in the first instance, but may extend to the mitral, and by preference to the anterior segment. The valves become swollen, and are soon studded with vegetations, consisting of perishable embryonic tissue, which quickly undergoes fatty metamorphosis. The valves are then softened, and may be torn by the pressure of the blood; they may undergo ulceration, or become the seat of aneurism. Abundant deposit of fibrin takes place upon the damaged valve; this may be subsequently wafted by the blood current into a remote vascular district, constituting arterial or capillary embolism, or it may undergo fatty change and disintegration, the molecular detritus so produced becoming a further source of blood poisoning. In the *débris* of these deposits M. Lancereaux has found parasitic forms resembling vibrios. The symptoms are those of septicæmia, and the disease runs a rapid course. On theoretical grounds, he suggests the use of phenic acid as the most appropriate treatment.

I have not met with a case of endocarditis traceable to the cause mentioned by Lancereaux. Examples of the disease of septic or pyæmic origin, are not, however, of unfrequent occurrence; but, as already stated, the inflammation is, in such cases, usually located on the right side of the heart.

With regard to the relative frequency of the several forms of acute inflammation of the heart, Dr. Stokes thinks endocarditis the most frequent; in the isolated form, however, he considers that pericarditis is of more frequent occurrence than endocarditis. Flint and Niemeyer hold the opposite opinion. No doubt if all, or even the majority of, the examples of valvular dis-

* *Archives Générales de Médecine*, Juin, 1873.

ease were admitted as evidence of antecedent *acute* endocarditis, the preponderance of this, in an isolated form, would be unquestionable. But at least one-half the cases of valvular lesion met with in practice are traceable to *chronic* inflammation, arising from strain, abuse of alcohol, or the rheumatic or gouty diathesis. It should be likewise borne in mind, that whilst secondary endocarditis, or chronic valvular disease with temporary excitement of the heart, may be readily mistaken for acute primary endocarditis, no such error is likely to be committed in regard to pericarditis, which, in most instances, involves adhesion of the pericardium in the first attack. Hence, a liability to over estimate the actual numerical proportion of acute primary endocarditis; whilst, in regard to pericarditis, the opposite error may be readily committed.

A further element of derangement is furnished by the occurrence, by no means infrequent, of a soft systolic murmur at the apex in the advanced stages of atonic rheumatism, or in the early period of convalescence, *not* from endocarditis, but from simple valvular inadequacy of a non-inflammatory character, and resulting, according to very high authority,* from ataxy of the papillary muscles. I believe, however, that the murmur in question arises from atony and partial yielding of the wall of the left ventricle at the acme of systole. Excluding all such cases as those just referred to, and admitting for comparison only examples of simple acute primary endocarditis on the one hand, and of simple acute primary pericarditis upon the other, I have no doubt the latter preponderate numerically.

Acute endocarditis is confined in extra-uterine life to the left side of the heart, cases of traumatic and septic origin being excepted; in the fœtus it is quite as strictly limited to the right side. The right chambers of the heart were primarily and exclusively affected in only 1 per cent. of the cases brought under the notice of the Pathological Society of Berlin, within the three years, 1868–71, inclusive. The tricuspid valves were alone engaged in the proportion of 1 per cent., and conjointly with other valves in the proportion of 10 per cent.; the pulmonary, in combination with other valves, 1 per cent.; the mitral alone, 52 per

* Niemeyer.

cent., in combination with other valves, 85 per cent.; the aortic alone, 13 per cent., in combination with other valves, 43 per cent.*

After birth, the contact of highly oxidized blood, and the strain to which the walls and valves are subjected in maintaining the arterial circulation, are supposed to furnish an adequate explanation of the greater proclivity of the left chambers to acute inflammatory action. On the other hand, I incline to attribute the greater susceptibility of the right chambers before birth, to the circumstance that, at this period of life, the oxidized blood from the placenta is passed in greatest part directly to the right side of the heart through the *ductus venosus* and ascending cava.

The valvular portion of the endocardium is that which is almost exclusively affected in endocarditis.

From the records of the Pathological Society of Berlin† it appears that, of 300 examples of endocarditis which had been exhibited in the three years above mentioned, inflammatory changes were confined to the valves in *every instance, except one*. The greater liability of the valves to the invasion of acute inflammatory disease may be accounted for, in part, by the concentration of the blood currents at the orifices, and the severe friction thence arising; but, chiefly by reference to the strain and tension to which the valves are subjected during ventricular systole. Taking this last mentioned circumstance into consideration, and referring only to the left side of the heart as the ordinary seat of endocarditis, an explanation may be thereby afforded of the much greater liability to attack, of the mitral than of the aortic valve. Indeed, so much greater is the liability of the mitral to acute invasion, that acute aortic valvulitis, save as a result of mechanical injury, must be regarded as exceedingly rare, and quite exceptional. Hence it may be assumed that the mitral valve is, *par excellence*, the seat of acute endocardial inflammation.

Of the pathogenesis of this affection in acute rheumatism, of which it constitutes so frequent and so grave a complication, two different theories have been propounded.

* *Edinburgh Medical Journal*, July, 1873.

† *Loco citat.*

Doctor B. W. Richardson attributes it to the direct contact of blood impregnated with lactic acid, the product of chemical changes in the lungs during respiration, and thence conducted by the pulmonary veins to the left side of the heart. The experiments upon which he bases this theory were performed upon dogs, cats, and rabbits, and consisted in the injection of a 10 per cent. solution of lactic acid into the peritoneum.* In these experiments the auriculo-ventricular valves were chiefly and almost exclusively engaged; the semilunar valves being only slightly thickened, but not sufficiently to interfere with their functions.

Doctor G. Reyher questions the validity of Richardson's conclusions, alleging that in several experiments performed by him on dogs, apparently healthy, a similar condition of the valves was found when death was caused by the injection of air into the jugular veins, or by poisoning with prussic acid. He urges that pathological changes in the heart-valves of dogs are of frequent occurrence idiopathically, and that the error of Richardson, and of Rauch, who, from independent experiments, arrived at the same conclusions, arose from their not having previously examined the hearts of healthy dogs. He concludes as follows: "In all the thirty-two apparently healthy dogs whose hearts were examined, the changes observed were identical with those described by Richardson and Rauch as the result of lactic acid injection.

"As Richardson and Rauch have not sufficiently avoided sources of error (owing to neglect of the examination of healthy dogs), their investigations are of no value in support of the supposed origin of endocarditis from the injection of lactic acid into the peritoneal cavity or jugular veins; consequently, that endocarditis arises from an accumulation of lactic acid in the blood remains *unproven*."†

In a communication of great practical value, Dr. Balthazar W. Foster, has recently furnished strong clinical evidence in sup-

* *An Experimental Inquiry on Endocarditis by the Synthetical Method*, London, 1353.

† *British and Foreign Medico-Chirurgical Review*, January, 1862, from *Virchow's Archiv.*, band xx.

port of the lactic acid theory of acute rheumatism.* Two men, suffering from diabetes mellitus, were put under the lactic acid treatment for that disease, as first recommended by Dr. Cantani, of Naples. In the first of these cases, no less than six successive invasions of acute articular rheumatism followed the use of the lactic acid in daily quantities varying from 3ij to ℥xx. The articular pain and inflammation subsided, in each instance, within twelve hours after the acid had been stopped, and they recurred with equal regularity within the same period after its use had been resumed. In the second case, one such attack followed the use of the acid, and promptly subsided when it was given up. Dr. Foster remarks: "The phenomena corresponded in all respects to those which are characteristic of acute articular rheumatism. They came on when the acid was taken, and ceased when it was discontinued. When moderate quantities of the acid were tolerated, an increase in the dose was succeeded by the painful inflammation of the joints.

"Coinciding with the development of the articular affection, was the appearance of perspiration, at first only slight, but afterwards, in the more severe attacks, copious and acid. These facts have dispelled the last lingering doubt in my mind as to the truth of the lactic acid theory of rheumatism."

According to Billroth and Weber, the blood of a person actually suffering from fever, of whatever kind, acts as an inflammatory irritant, and the endocardium is in a preeminent degree liable to inflammation from such cause.

The period of an attack of acute rheumatism at which endocarditis is most frequently manifested, is from the sixth to the ninth day, according to my observation. Fuller extends the period from the sixth to the twentieth day. Pleuritis and pneumonia, though frequently associated with pericarditis and endopericarditis, are of comparatively rare occurrence in connexion with simple endocarditis. According to Fuller's statistics, one or other form of acute pulmonary inflammation existed in the former connexions in the proportion of 1 : 1·7, and 1 : 1·4 respectively, and in the latter only in the ratio of 1 : 10.

* *British Medical Journal*, December 23rd, 1871, and *Clinical Medicine*, 1874, p. 144.

Is pulmonary inflammation, in such cases, ever in reality the primary disease, endocarditis being secondary to, or dependent upon it? Niemeyer is of opinion that such is the case, though very rarely. I have not met with a single example of the kind, out of the many hundreds of cases of acute pulmonary inflammation which have come under my notice during the past eleven years, since I became connected as physician with a large hospital.

Owing to the fact that acute endocarditis is not fatal in the initial stage, the opportunity for determining the structural alterations attendant upon it in man has fallen to the lot of very few observers; and the doctrine held upon this subject is the result, mainly, of induction from experiment upon inferior animals.

Richardson states that when the animals experimented upon by him were examined within a period of ten hours after the introduction of a solution of lactic acid into the peritoneum, the tricuspid valve was found to be highly vascular and villous, to have lost its ordinary polish and transparency, and to have exhibited minute droplets of lymph upon the free margin. Examined at a somewhat later period, the segments were tumid, and inadequate to close the orifice, and yielded an opaque and viscid exudation on being punctured. At a still more advanced period, they were dense and thickened, but less swollen, and exhibited beneath the surface a layer of solid fibrin, and masses of the same material deposited upon the edges. Finally, they were found to have shrunk and become inadequate by retraction of their edges. With regard to the genesis of the tissue-changes which characterize the several stages of acute valvular inflammation, the first is distinguished by congestion of the *vasa vasorum*, proliferation of the connective tissue corpuscles, and consequent thickening and opacity of the valve-curtains. The second stage consists in efflorescence, or outgrowth on one of the lamellæ, by accumulation of corpuscular elements, which are liable to undergo granular metamorphosis, and subsequent disintegration and detachment under the action of the blood-current. Hence so called ulceration of the valve, if one lamella only be involved, or perforation if both lamellæ are eroded.

Upon the corpuscular outgrowth previous to disintegration, or upon the jagged edges of the ulcer or perforation caused by this process when it has actually occurred, fibrin is precipitated from the blood, forming rugosities or vegetations, which, in their turn, are liable to undergo granular metamorphosis and subsequent disintegration under the friction of the blood-stream. Virchow, however, denies entirely the occurrence of fibrinous precipitation, and attributes the primary changes of structure to corpuscular proliferation exclusively; a view which is unreservedly adopted by Niemeyer.

Other changes may follow the primary ulceration of the valves. Thus, valvular aneurism may result from the entrance of blood through the ulcerated aperture in one lamella, and the gradual detachment and distension of the other into the form of a pouch; or, a still more exceptional occurrence, veritable supuration may take place, giving rise to small abscesses, not larger than a pin's head. Even further consequences may ensue: softening and rupture of the chordæ tendineæ (Law, Niemeyer); laceration or ulceration of the endocardium at other points of its surface, leading to the "acute consecutive aneurism" of Breschet, or the "acute aneurism" of Rokitansky, if the ventricular wall be likewise softened by extension of the inflammatory process; adhesion of the valve-segments to one another, or to the wall of the ventricle or artery as the case may be; or cohesion and fusion of the chordæ tendineæ.

Agglutination of the valves and tendinous chords by acute inflammation is, no doubt, the cause of that peculiar form of auriculo-ventricular stenosis known as "the funnel-shaped" aperture. Embolism and consequent hæmorrhagic infarction, or metastatic abscess in the spleen, the kidneys, the liver, the lungs; or, in the brain, hæmorrhagic foci (capillary apoplexy), localized anæmia and softening of the substance beyond, are possible accidents of earlier occurrence.

An immediately fatal result is, however, very seldom witnessed in acute rheumatic endo- or pericarditis. I have not met with a single example of the kind; and, of 71 cases, only 3 were immediately fatal in the experience of Dr. Peacock. In forming a prognosis, therefore, the remote consequences are mainly,

and all but exclusively, to be considered. These are of the gravest character, and may be included under the three heads of valvular obstruction, valvular inadequacy, and consecutive changes in the walls of the ventricles or auricles.

The *symptoms* of acute inflammation of the endocardium, where the muscular substance of the heart is not in some degree involved, a contingency of rare and exceptional occurrence, are by no means characteristic. This is due to the fact that they are but imperfectly localized, and, being of a general and febrile character, they are masked by those of the acute fever which constitutes the primary and the principal disease. The patient, however, not unfrequently refers to the region of the heart as the special seat of peculiar discomfort and distress. The breathing is in some degree quickened; pressure over the heart is not well borne; its action is somewhat tumultuous, and decubitus on the left side is avoided.

Even these symptoms are occasionally absent; so that the physical evidence affords the only positive information of the presence of endocarditis. This consists in the occurrence with the first sound of the heart of a murmur which, in the majority of instances, is located at the apex, and but very rarely heard at the base.

Bouillaud and Skoda lay much stress upon the extension of precordial dulness, associated with the symptoms of engorgement of the right side of the heart, as evidence of endocarditis. Such a phenomenon is, no doubt, occasionally witnessed, but only where the muscular substance of the heart is likewise deeply and seriously involved; or where, without this complication, engorgement of the right chambers arises from pulmonary emphysema with intercurrent bronchitis. Is systolic murmur always present where endocarditis exists? And, when actually present in a case of acute rheumatism, should it be regarded as proof of the existence of endocarditis? To the former question I venture to give an affirmative but qualified answer, and to the latter, a negative.

It has repeatedly happened to me, in making my daily examination of the heart in cases of acute rheumatism, to find a faint, blowing, systolic murmur at the apex, which, by many, would be

regarded rather as a muffling or prolongation of the first sound, where no symptoms or signs of cardiac complication had existed on the previous day. Most frequently this has coincided with one or more of the symptoms already mentioned as referable to the heart; but occasionally, though rarely, it has been unattended by any of these. On the following, or on the second day, this faint buzz has developed into a veritable bellows-murmur accompanying the first sound. I may add that, the adynamic murmurs of convalescence alone excepted, in no instance has it occurred to me to discover in the progress of rheumatism a fully developed murmur suddenly announced, where no trace of its nascency had previously existed; or where the coexistence of a loud pericardial frottement may not have masked it at that period of its development.

Doctor Stokes is of opinion, that in the ~~state of~~ typhoid debility not unfrequently associated with endocarditis, murmur may be suppressed or suspended. No doubt, in the profound debility preceding dissolution this may be the case; but, with this exception, I have not met with an example of complete suppression of murmur, where endocarditis was subsequently proved to have existed at the time, either by the development of a well pronounced murmur, or by *post mortem* evidence.

The coexistence of pericardial effusion may suppress an endocardial murmur; but the positive signs of the presence of liquid would forbid a negative diagnosis in regard to endocarditis, where murmur had not been previously detected.

Finally, the friction-sound of a concomitant pericarditis may completely mask an endocardial murmur, more especially at an early period, when the latter is not yet well pronounced. In the progress of the compound disease, the friction-sound will be definitively suppressed by cohesion of the opposed surfaces of the pericardium, whilst the valvular murmur will be heard, perhaps for the first time, in its complete intensity. Thus, the signs of pericarditis and of endocarditis proceed, as to development, in opposite directions; the former towards extinction, and the latter towards maximum intensity. But the circumstances above indicated would not warrant the inference that endocarditis had

existed without murmur; they would only show that it had attained a certain development without *audible* murmur.

The *associations* of a systolic apex-murmur in acute endocarditis, are of great importance in regard to differential diagnosis and the prospects of the patient. A murmur, *accompanied* by a first sound, and a regular though quick and weak pulse, indicates simple endocarditis; but, *replacing* the first sound, and especially if attended with a failing and irregular pulse, such a murmur should be regarded as diagnostic of endo-myocarditis. The weakness of the left ventricle, consequent upon inflammation of its muscular wall, serves to explain both the irregularity of the pulse and the suppression of the first sound; the valve element of this sound being abolished by the inflammatory thickening and softening of the mitral curtains, and the impulse element by the debility of the ventricle, consequent upon similar changes in the myocardium.

In acute inflammatory fever, the existence of an endocardial murmur must not be regarded as proof of endocarditis. It may be anæmic; and, if localized at the base of the heart, especially at the left base, it is most probably of this character, and a careful scrutiny should be instituted with a view to determining the question. The absence of *intrinsic* murmur in the carotid arteries, *i.e.*, when pressure is not made with the stethoscope sufficient to develop a murmur in these vessels; a venous hum in the jugulars; and the diminution of intensity of the endocardial murmur, or its complete suspension, on placing the patient in the sitting posture, may be regarded as proof that the murmur is anæmic. If the patient be a young female, the foregoing evidence in favour of this view is thereby strengthened; but even in the absence of this circumstance it is sufficient for the positive diagnosis of hæmic murmur. The murmur may be due to chronic valvular disease, or to this condition with secondary acute endocarditis. In the former case, the outward displacement of the apex, and the absence of symptoms of recent acute inflammation of the heart, constitute sufficient diagnostic evidence, even where the ordinary consequences of chronic inadequacy of the mitral valve do not exist; namely, pulmonary and general venous congestion, with dropsy.

The supervention of acute endocarditis on chronic valvular disease is more difficult of diagnosis; indeed, it is scarcely recognizable with certainty. The symptoms of a recent accession of acute inflammation of the heart, more especially increased embarrassment of breathing, may warrant a presumptive diagnosis; but, the physical signs being already appropriated by the chronic disease, a more positive opinion cannot be ventured. The subsequent course of the illness will, however, remove this difficulty. All the symptoms and signs of the chronic lesion are aggravated by the products of the recent affection.

Acute endocarditis is very rarely, if ever, especially in those of intemperate habits, unaccompanied by inflammation of the substance of the heart to a greater or less extent; and in proportion to the depth to which the muscular substance is involved, the symptoms assume a more formidable character. To this circumstance is due the special value of the symptoms, as distinguished from the signs, of acute endocarditis. The failure of the pulse, the restlessness and dyspnoea, the engorgement of the right chambers, rapid enlargement of the liver, congestion of the kidneys, œdema of the lungs, and the general dropsy, which occasionally supervene within a brief period, are due, not to endocarditis, but to its complication, myocarditis. The muscular substance of the heart, to the extent to which it has been implicated in the inflammatory process, will, if examined shortly after the acute attack, be found sodden and of a dead-leaf tint. At a more remote period, it exhibits the tissue-changes characteristic of fatty degeneration. Hence, whether viewed in regard to attendant symptoms, or remote consequences, the gravity of endocarditis should be measured by the extent to which the muscular substance of the heart is judged to be involved, rather than by the degree of inflammation of its lining membrane. Even in the most favourable cases, however, where the inflammation is strictly confined to the endocardium, representing but a small percentage of the total number, the remote prospect for the patient is eminently discouraging. The valves are thickened and in the first stage of disorganization, whether by morbid development of their normal elementary structures, or by deposit from the blood. Hence, retrogressive changes of structure, in-

volving proportionate incompetency of the valves, or obstruction from their morbid growth.

In the opinion of some writers, a process of tissue-change in the direction of cure may be set up; the hypertrophied connective tissue of the valves being reduced to its normal proportions by absorption, and the deposit, if any, disintegrated and detached by the blood current, to be subsequently dissolved and excreted. I cannot subscribe this doctrine, inviting though it be. The valves of the heart are not so circumstanced as to admit of this favourable change. They are, in the performance of their functions, subject to the pernicious influence of three forces operating alternately; namely, motion, friction, and tension, which are eminently calculated to maintain in them a state of constant irritation or chronic endocarditis, necessarily resulting in progressive valvular disorganization. This downward progress, once set up, may be restrained or retarded, but not arrested or reversed.

Chronic endocarditis is the result of antecedent acute inflammation of the endocardium, which leaves some thickening of the valves in the line of their mutual contact. This thickening, under the continued irritation arising from motion, tension, and blood-friction, increases by inflammatory outgrowth of the connective-tissue corpuscles, and subsequently, having undergone condensation and contraction, may become calcified; or, more rarely, it may pass through the regressive stages of fatty change and disintegration. Hence, extreme disfigurement, rather than destruction of the valves, is the ordinary result of chronic endocarditis.

In chronic endocarditis, the muscular substance, if the inflammation have extended to it, may be replaced by fibroid tissue; and this, being less resisting, may yield and give rise to a "true aneurism," the "chronic aneurism" of Rokitsansky.

In the *treatment* of acute endocarditis, I proceed upon the principle of moderate local depletion, followed by mild but rapid mercurial action. Blood is drawn cautiously by means of leeches applied to the precordium; and from day to day this process is repeated, till the urgent symptoms of dyspnoea and precordial oppression are alleviated or subdued, the leeches

being succeeded by warm cataplasms over the heart. I have no fear of increasing the debility of the patient by these measures, deeming the prompt and complete arrest of inflammatory action, as far as this can be accomplished, the prime consideration. In view, however, of the necessarily protracted illness of the patient, I deprecate vigorous depletion, and am satisfied with leeching, being guided as to the quantity of blood drawn, and repetition of procedure, by the constitution, age, and actual strength of the patient. The pulse is usually increased in volume and diminished in rate under this treatment, whilst respiratory distress and cardiac pain or oppression are alleviated. Mercury is then administered rapidly, but in minute doses, to the point of producing its earliest results; namely, foetor, and a metallic taste. Half a grain of calomel with a grain of James' powder is given every half hour, or every hour, according to the urgency of the case, as first suggested and practised by Dr. Law; whilst a plaster, composed of half an ounce of emplastrum hydrag. and ten grains of opium, is laid over the heart, as recommended by Dr. Beale. It will be seen that I eschew the heroic depletion of Bouillaud upon the one hand, and the copious exhibition of mercury recommended by Hope, on the other. Such extreme measures, directed to an object in my judgment unattainable, namely, the complete subdual of inflammation of the valves, or the dispersion of their endogenous outgrowths, are not warranted by even the most favourable prospects from treatment.

Whilst discountenancing the heroic line of procedure just noticed, I can by no means subscribe the sceptical doctrine of Niemeyer in regard to the alleged inefficacy of all modes of treatment. I believe that whilst it is eminently irrational to propose to one's self a complete cure of endocarditis by any medicinal agency, arrest of the process of inflammation, and restriction of its products within limits compatible with partial restoration to health, and many years of life, is attainable by the plan above proposed. Examples confirmatory of this statement will be found among the succeeding cases. To allay nervous excitement, and procure sleep, an opiate should be given at night; *e.g.*, fifteen to twenty minims of Battley's sedative, or a grain to a grain and a-half of the aqueous extract of opium. Subsequently, when the acute

symptoms have subsided, tonics should be administered; and of these I give a preference to quinine and strychnia, with or without iron, as being, in my judgment, the most efficacious. During the treatment, and long subsequently, complete repose of body and mind should be strictly enjoined. Alcoholic stimulants of the stronger kind should be prohibited; but good claret and mild bitter beer may be allowed in moderate quantity. The diet should be nutritious and concentrated; excess of liquids, whether as food or drink, being inadmissible, owing to their effect in temporarily increasing the volume of the blood, and thereby increasing the strain upon the walls and valves of the heart.

The following cases are presented as examples of endocarditis in its simple and in its complicated forms:

CASE LXXIV.—*Acute Rheumatic Endo-Myocarditis; Recovery.*

Mrs. C., aged about twenty years, was visited by me, in consultation with Mr. Cahill of Dame-street, June 20th, 1871. She had an attack of rheumatism some years ago, and a second at Christmas, 1870. Ten days previous to my visit she had a third attack; the muscles of the chest and the joints of the hands being the seat of inflammation. On the eighth day of illness, dyspnoea with cough set in, and, on the following morning, Mr. Cahill diagnosed endocarditis. When visited by me, she complained chiefly of weakness, and a feeling of oppression at the precordium. There was no swelling of any of the joints; and the muscular pains had been alleviated by the free use of a liniment, of aconite and laudanum, and a flannel swathe, previously directed by Mr. Cahill. She was perspiring freely; respiration was laboured and accelerated; pulse very small and weak, 120 in the minute, but regular. There was slight cough without expectoration; the urine was scanty, and deposited lithates in large quantity. Tongue moist and coated; decubitus indifferent; no vertigo, delirium, or other head symptom. The bowels were rather relaxed, and she complained of sore throat; but there was no external swelling of the neck. The action of the heart was rather tumultuous; precordial dulness not extended; apex-beat

in the normal position, and here a very soft bellows-murmur replaced the first sound; this murmur was likewise audible in the left axilla, but not elsewhere. At the base, and in the course of the aorta, the sounds of the heart were normal, and no *frémissement* anywhere existed. The base of the right lung was dull, and yielded mucous râles. Ordered: Four leeches to precordium, to be followed by a mercurial and opiate plaster. Two grains of grey powder, and two of Dover's powder to be given every second hour; chest to be dry-cupped over right base, and then wrapped in cotton wadding. A small glass of champagne to be given every second hour. Liquid food. Chloral and opiate draught at night. I did not visit this lady subsequently, but I learned from Mr. Cahill that she made a good recovery.

Remarks: Here, as in all cases of acute endocarditis where the muscular substance of the heart is deeply engaged, the murmur of mitral reflux was not only soft and blowing, but *substitutive*; the impulse element of the first sound being entirely suspended by the softening and debility of the muscular substance of the heart produced by inflammation, and the valve element muffled by thickening, and masked by the eddy of reflux. That the implication of the heart was of recent date, and most probably coeval with the last attack of rheumatism, is shown by the normal position of the apex, and by the cardiac symptoms actually present at the date of examination.

CASE LXXV.—*Acute Rheumatic Pericarditis, succeeded four months later by Acute Endo-Myocarditis; Death. Autopsy: Hypertrophy of the Heart; General Adhesion, with Infiltration of the Pericardium; Thickening, Opacity, and Incompetence of the Mitral Valve; Fatty Transformation.*

Margaret O'B., aged thirteen years, under my care for scarlatina in December, 1872, was admitted into hospital on Easter Monday, 1873, under my colleague, Dr. Nixon, for articular rheumatism with pericarditis, from which she made a slow and incomplete recovery.

She was re-admitted on the 18th August following, in a state of debility, with dyspnoea and palpitation; the feet were slightly swollen.

About the end of August she complained of sharp pain in the region of the heart, accompanied with great tenderness, and gasping respiration. A systolic murmur, blowing and substitutive, was now audible at the apex, and faintly in the left back. The pulse became excessively weak, and also irregular and intermittent; varying as to rate from 108 to 120 in the minute. Respiration 72. There was extreme restlessness, accompanied with frequent moaning, and inability to lie down. The lips were livid, the pupils dilated, and the extremities cold. The liver was greatly enlarged. The impulse of the heart was feeble and tumultuous, and precordial dulness was much extended generally. The lower limbs became in the highest degree œdematous, and required puncturing; this was practised with great relief to the tense integument, and was followed by copious discharge of serum. Signs of pulmonary œdema and hydrothorax now appeared; the latter apparently kept in check by the free discharge of serum from the punctures in the feet and legs.

During the last week of life her condition was most pitiable; the slightest movement of her body was followed by painful palpitation, and extreme respiratory distress, accompanied with moaning. For several days preceding her death, which took place on the 15th September, there was scarcely any pulsation to be felt in the radial arteries.

The body was examined on the day of demise. There was a good deal of serum in the cavities of the pleuræ; the bases of both lungs were congested; the pericardium was universally adherent to the heart, the adventitious connective tissue which united the two surfaces, being infiltrated with lymph and serum, and fully half an inch thick at the base of the heart. It presented, in a few situations, masses of yellow plastic fibrin about a quarter of an inch in diameter, and containing areolæ filled with clear serum. Examined under the microscope, this neoplasm presented the histological characters of white fibrous tissue, and disappeared under the action of strong acetic acid. The heart, with the pericardium attached, weighed fourteen ounces; the walls of the left ventricle were thickened, and its cavity somewhat dilated; both segments of the mitral valve were greatly thickened at the edges by sub-endothelial formation, and

were incompetent; and both the valves and the adjacent endocardial lining of the ventricle were opaque. The other cavities and valves were normal. The muscular substance of the left ventricle was of a dun-brown tint; and, examined under the microscope, it nowhere exhibited a trace of striation. The fibrils were represented by parallel lines of minute oil dots, disposed in the length of the fibre. Several larger oil drops were observed free between the fibres.

I conclude that adhesion of the pericardium had existed during a period of at least four months prior to the last and fatal illness; and that this illness had consisted in general carditis, producing its specific effects in each of the tissues involved; viz., thickening and opacity, with incompetency of the mitral valve; dinginess of hue and fatty metamorphosis of the muscular substance, and fibro-serous infiltration, with softening and thickening of the pseudo-membrane between the heart and pericardium. The diagnosis of general carditis was made immediately after the access of the last acute attack, from the great respiratory and cardiac distress and failure of the pulse; but that of adhesion of the pericardium was only made presumptively. The case affords an example of serous effusion into the pericardium by infiltration of old adhesions.

CASE LXXVI.—*Endo-Myocarditis; Recovery.*

On the 1st January, 1873, I was summoned to visit Miss F., aged six years, of delicate constitution, and subject to anomalous pains in the limbs, supposed by the parents to be "growing pains;" but which, as they were generally associated with a febrile condition and a deposit of lithates perceptible in the urine, I inclined to regard as rheumatic.

For some days previous to my visit, she had complained more than usual of these pains, and likewise of uneasiness in the region of the heart. I found her in a feverish condition, with rapid but regular pulse, and quick breathing. There was precordial tenderness; the action of the heart was tumultuous, but the sounds were clear, and unassociated with murmur. In the course of a few days the pulse became irregular, and very weak, whilst the

first sound of the heart was replaced by a loud bellows-murmur. The breathing was very rapid, and there was a tendency to syncope, with frequent sighing and loss of sleep.

This child's condition was, for several weeks, most precarious; but she made a good recovery under the use of small doses of digitalis, iron, and spirit of nitrous ether. She has since become very fat, and is able to take moderate exercise without inconvenience; but she is readily put out of breath by rapid walking, or ascent of stairs. On examining her heart ten months later, I found a loud systolic bellows-murmur *accompanying* the first sound at the apex. The pulse was quite regular, and the general condition was that of good health.

The points of interest in this case are the irregularity of pulse, and a tendency to syncope, coincident with the development of a *substitutive* systolic murmur at the apex of the heart; and, on recovery, the restoration of the first sound *accompanied* with a murmur.

Valvular disease of the heart presents a most striking illustration of functional disability from mechanical impediment. The valves placed at the auriculo-ventricular orifices, and at those of the aorta and the pulmonary artery, for the purpose of imparting a definite direction to the blood-current, by preventing its retrograde movement during the systole of the ventricles and of the arteries respectively, may, by having become thickened, tuberculated, or otherwise disorganized, oppose the passage of the blood onwards in its normal course, or permit it to flow backwards into the chamber whence it had been expelled by the preceding systole. Hence, valvular diseases, functionally considered, are reducible to two generic forms, *obstructive* and *regurgitant*. Anatomically regarded, they should be classified as auriculo-ventricular, and arterial. It is more convenient, however, to designate valvular diseases specifically, as aortic, mitral, pulmonic, and tricuspid, respectively. If, to these latter designations, be appended those which imply the functional derangements actually present, a sufficiently precise nosological classification will have been attained. Thus regarded, all valvular diseases of the heart might be classified as follows, viz. :

Aortic	{ (a) Obstructive. (b) Regurgitant.
Mitral	{ (a) Obstructive. (b) Regurgitant.
Pulmonic	{ (a) Obstructive. (b) Regurgitant.
Tricuspid	{ (a) Obstructive. (b) Regurgitant.

But obstruction and regurgitation may be due to constriction and dilatation of the several orifices, irrespectively of the state of the valves. From direct physical evidence alone, it is difficult, and in the opinion of most writers impossible, to distinguish lesion of the orifice from lesion of the valve.

Sir W. Gull declares that "auscultation alone cannot determine whether what has been called a mitral murmur, results from organic or functional change."*

Between these two causes of murmur, at least in regard to those dependent upon regurgitation, the differential diagnosis is, however, in a great degree practicable. It is based upon the coexistence of the sound of valve-tension, and in part upon the peculiar rhythm of the murmur, when inadequacy results from dilatation of the orifice mainly or exclusively. When the valvular element of sound is associated with murmur at any of the orifices of the heart, it may be legitimately inferred that the valves appertaining to that orifice are either sound, or but partially disorganized. If, further, the murmur be posterior to the sound in time, it may, with confidence, be affirmed that there is only a partial leakage at the orifice; and that this arises either from a trivial, and most probably a recent, lesion of the valve, or from dilatation of the orifice exclusively.

Numerous examples, confirmatory of the foregoing statement, will be found among the cases appended to the sections on aortic and mitral disease.

Valvular disease of the heart is, with the single exception of phthisis, the most prevalent form of organic disease in the United Kingdom.

On the authority of Dr. A. W. Barclay,† out of a total of 419

* *Guy's Hospital Reports*, vol. iv., 1846, proposition 38.

† *Medico-Chirurgical Transactions*, vol. xxxi., 1848.

bodies examined in St. George's Hospital during the years 1846 and 1847, with reference to the state of the circulatory organs, 79, or a proportion of 18·8 per cent. exhibited valvular lesion of some kind. The relative proportion of aortic and mitral valve disease, single or combined, amongst these 79 cases, was as follows :

Aortic and Mitral, 36, or 45 per cent. = 8·6 per cent. of total cases.					
Aortic alone,	26,	„	33	„	= 6·2 „
Mitral alone,	17,	„	22	„	= 4·0 „
	79		100		18·8

Of 367 cases of valvular disease, collected by Dr. King Chambers from the records of St. George's Hospital, the mitral and aortic valves were affected in 121; the aortic valves alone in 107; the mitral alone in 96; mitral and tricuspid, 10; mitral aortic and tricuspid, 10; all four sets of valves, 9; tricuspid alone, 1; tricuspid and aortic, 2; aortic, mitral, and pulmonic, 2; aortic and pulmonic, 4.*

Doctor Flint states that, in a total of 271 examples collected by him, the mitral valve was affected in 111, and the aortic in 72. In 66 of these, both the mitral and aortic were engaged. In 14 instances only were the tricuspid or pulmonic valves affected.†

My clinical records include many hundred cases of valvular disease of the heart; but, the pathological returns at my disposal, upon which alone I propose to base my calculations, furnish only 44 examples of valvular lesion. In each of these, the body was carefully examined after death by myself personally, with the aid of my clinical clerks. The following table (X.) exhibits in summary the results obtained.

* Decennium Pathologicum, *British and Foreign Medico-Chirurgical Review*, vol. xii., 1853; and *Medical Times and Gazette*, July to December, 1852.

† *Diseases of the Heart*, second edition, 1870, p. 129.

An analysis of the foregoing table yields the following general results:

Total number of Cases, 44: males 28 (= 63·63 per cent.); females 16 (= 36·36 per cent.) Average age, thirty-seven years and four months.

					% of total cases.
The aortic valves were engaged	in 26	= 59·09
causing obstruction alone	9	= 20·45
regurgitation alone	4	= 9·09
both obstruction and regurgitation	13	= 29·54
The mitral valves were engaged	29	= 65·90
causing obstruction alone	4	= 9·09
regurgitation alone	13	= 29·54
obstruction and regurgitation	12	= 27·26
The tricuspid valves were engaged	5	= 11·36
causing obstruction alone	1	= 2·27
regurgitation alone	2	= 4·54
obstruction and regurgitation	2	= 4·54
The pulmonic valves were engaged	1	= 2·27
causing obstruction (a cancerous nodule) alone	1	= 2·27
Aortic obstruction was associated with mitral regurgitation	8	= 18·18
Mitral obstruction was associated with tricuspid regurgitation	11	= 25·00
Mitral obstruction and regurgitation, with aortic obstruction and regurgitation	2	= 4·54
with tricuspid obstruction	2	= 4·54
with tricuspid obstruction and regurgitation	1	= 2·27
Hypertrophy of the right ventricle was present	14	= 31·81
Hypertrophy of the left ventricle	33	= 75·00
Dilatation of the right ventricle	24	= 54·50
Dilatation of the left ventricle	24	= 54·50
Fatty degeneration of the heart was found	20	= 45·45
associated with aortic valve disease	14	= 31·81
viz., aortic obstruction	6	= 13·63
aortic regurgitation	3	= 6·81
both obstruction and regurgitation	5	= 11·36
Renal disease existed	7	= 15·90
associated with aortic valve disease	5	= 11·36
viz., aortic obstruction	4	= 9·09
aortic obstruction and regurgitation	1	= 2·27
associated with mitral valve disease	4	= 9·09
viz., mitral obstruction and regurgitation	2	= 4·54
mitral regurgitation	2	= 4·54
associated with hypertrophy of the left ventricle	7	= 15·90
of the right ventricle	1	= 2·27
of both ventricles	1	= 2·27

Renal disease (<i>con.</i>)									
associated with dilatation of the left ventricle	in	3	=	6.81	% of total cases.	
of the right ventricle	"	4	=	9.09	"	
of both ventricles	"	2	=	4.54	"	
Thrombosis of the right ventricle existed	"	36	=	81.81	"	
of the left ventricle	"	12	=	27.26	"	
Atheroma of the aorta was present	"	22	=	50.00	"	
associated with renal disease	"	2	=	4.54	"	
with fatty degeneration	"	18	=	29.54	"	
with disease of the aortic valves	"	16	=	36.36	"	
with disease of the mitral valves	"	14	=	31.81	"	
Sudden (instantaneous) death occurred	"	12	=	27.26	"	
the valvular lesion was aortic	"	8	=	18.18	"	
viz., obstructive	"	3	=	6.81	"	
regurgitative	"	2	=	4.54	"	
obstructive and regurgitative	"	3	=	6.81	"	
The heart was fatty	"	6	=	13.63	"	
The aorta was atheromatous	"	6	=	13.63	"	
heart fatty, and aorta atheromatous	"	5	=	11.36	"	
heart fatty, aorta atheromatous, and aortic valves						
obstructive	"	3	=	6.81	"	
heart fatty, aorta atheromatous, and aortic valves						
inadequate	"	2	=	4.54	"	

The foregoing statistics may be considered to possess some value, as a contribution towards the settlement of certain debated questions in connexion with valvular disease of the heart. To these questions I shall have to revert in the progress of this chapter.

Valvular diseases of the heart are met with at every period of life; but, they are certainly more prevalent in middle age than in youth or senility. Under the title of "malformations," analogous conditions, as regards their mechanical effects, exist congenitally. These have been already considered (see p. 775). Valvular disease is likewise pretty equally apportioned between the sexes; although certain forms of it are more prevalent amongst males, and others amongst females. As to origin and rate of progress, it may commence with acute inflammation, and run its course rapidly, or slowly, according to the injury done to the mechanism of the heart in the first instance, and the circumstances in which the patient is subsequently placed, in regard to nutrition, and to physical and mental exertion. If the degree in which the valves have been structurally injured and functionally

impaired by the primary disease be but trivial, and if the mind and body be allowed adequate repose until the heart shall have adapted itself, by a gradual process of dynamical adjustment, to the altered conditions under which it is required to function, little if any inconvenience may be experienced by the patient during a life of average length and activity, as long as its nutrition is proportionately maintained. If, on the other hand, the circumstances be in all respects the opposite of those indicated, the downward progress will be rapid, in proportion as the adverse influences mentioned shall have been in operation. Irrespective of these considerations, certain habits and diathetic proclivities exercise a most deleterious influence upon valvular disease; especially alcoholic intemperance, and the gouty diathesis. But valvular disease may commence insidiously from habitual over-distension of the heart, and have made considerable progress before the patient has become sensible of its presence from notable inconvenience of any kind. This is especially true of mechanics and operatives engaged in any labour requiring great muscular effort. Habits of intemperance are capable of aggravating, in an especial manner, the evil consequences of such avocations; and, unfortunately, they are of common occurrence. The great fatigue which these men undergo, the copious perspiration caused by their labour, and the high wages they are able to earn, supply, at once, the excuse for intemperance, and the means of indulging in it.

By habitual saturation with alcohol, the blood acquires irritant properties; whilst the valves, by the repeated strain to which they are subjected, are predisposed to irritation. Under this twofold influence, sub-acute inflammation of the connective tissue of the valves, and abundant cell-growth, are prone to occur. Hence valvular thickening, obstruction, and inadequacy. Hence, likewise, corrugation, brittleness, and rupture or laceration of the valve-segments; ulceration of their surface; and deposit of fibrin by entanglement, constituting so-called vegetations.

Corvisart was of opinion that these growths are occasionally of syphilitic origin.* Hope maintained that in many cases they proceeded from a combination of syphilis and mercury.†

* *Opus citat.*, p. 194.

† *Opus citat.*, p. 367.

Laennec regarded them as organized fibrinous concretions, a view which Hope warmly contests, on the erroneous assumption that valvular concretions are analogous to polypi of the cavities. I have already (p. 800) shown that valvular excrescences are due primarily to cell-proliferation and upgrowth, supplemented by precipitation of fibrin upon the roughened surface of the valve. The fibrin so deposited may be promptly detached by the passing current and impacted in a distant artery, constituting an embolus; or it may undergo organization and be thereby incorporated with the valve, becoming subsequently degraded, under the influence of perverted nutrition, into one of the lower forms of tissue; viz., fat, fibro-cartilage, or calcareous nodule. Kreysig had attributed these growths to inflammation, in which opinion he was supported by Bertin and Bouillaud. Hope likewise adopted this view; adding, that he had produced them, in the course of an hour, by lacerating the pulmonic valves in an ass poisoned with woorara.* He believed that fibrin was precipitated from the passing blood-stream, through a certain influence exercised upon it by contact with an inflamed surface. Mere roughening of the surface is, however, in his opinion, competent to produce precipitation.†

Rupture, or laceration, of the valves is the most formidable lesion to which they are liable; because of the exceptionally severe character of the symptoms to which it gives rise, and the proximate death of the patient to which it infallibly leads. No less rapidly fatal is acute valvular inadequacy by rupture of the papillary muscles or tendinous chords. I believe, however, that neither accident can occur, unless the valves or their appendages have been structurally deteriorated by antecedent disease.

Corvisart was wrong in assigning greater frequency of occurrence to disease of the aortic than of the mitral valves.‡ Laennec, whilst expressing the opposite opinion, declared that, by a singular coincidence, he had witnessed at Corvisart's clinique, within a period of three years, more numerous examples of advanced ossification of the aortic valves than had come under his notice

* *The Diseases of the Heart and Great Vessels*, third edition, 1839, p. 370.

† *Ibid.*

‡ *Opus citat.*, section iv., p. 186.

in the following twenty years.* No doubt, special circumstances are capable of influencing the relative frequency of certain forms of valvular disease; even amongst civil communities. This remark applies in an especial manner to disease of the aortic valves, now generally recognized as affecting commercial populations in an excessive degree. Hence, most probably it is, that amongst the population of Liverpool, Dr. Waters has met with disease of the aortic valves more frequently than any other form of valve-lesion.†

As to relative frequency, I believe valvular diseases, in the isolated form, may be arranged in the following order :

- Mitral regurgitative.
- Aortic obstructive.
- Aortic regurgitative.
- Mitral obstructive.
- Tricuspid regurgitative.
- Tricuspid obstructive.
- Pulmonic obstructive.
- Pulmonic regurgitative.

In combination, their relative frequency is as follows :

- Aortic obstructive and regurgitative.
- Mitral obstructive and regurgitative.
- Mitral obstructive and tricuspid regurgitative.
- Aortic obstructive and mitral regurgitative.
- Mitral obstructive and regurgitative, with aortic obstructive and regurgitative.
- Mitral obstructive and regurgitative, with tricuspid obstructive.
- Mitral obstructive and regurgitative, with tricuspid obstructive and regurgitative.
- Aortic obstructive and pulmonic obstructive.

Bichat committed an egregious error in denying the occurrence of valvular disease on the right side of the heart. It is, however, beyond all comparison less frequent on the right than on the left side.

In my returns (Table X.), it is as 5 : 43. Hope estimates the proportion as 1 : 16 or 20.

* *Opus citat.*, tom. ii., p. 586.

† *Diseases of the Chest*, 1873, second edition.

According to Bertin,* this preponderance is due to the more irritant properties of arterial as compared with venous blood, which implies that, in his opinion, it proceeds from the greater liability of the left than of the right side of the heart to the invasion of endocarditis. This doctrine cannot be questioned, and the fact may be explained quite irrespectively of the qualities of the blood, by the greater strain or tension to which the left valves are habitually subjected, as long since remarked by Corvisart.†

The heart is capable of accommodating itself, *miro modo*, to slow and progressive change of the valves, leading even to extreme disorganization, consistently with the maintenance of an adequate circulation; provided only that the duty imposed upon it be not excessive. An example strikingly illustrative of the truth of this remark is given by Dr. Stokes.‡ A gentleman of middle age and active habits, who, up to a few days before his death had enjoyed uninterrupted good health, was attacked with rigors, followed by febrile symptoms, and attended with bronchial irritation. The heart pulsated with great violence, and a systolic bellows-murmur was heard. He died suddenly, one week after the date of his first illness. The left ventricle was found greatly distended with fluid blood, and the aortic orifice was contracted to a degree which Dr. Stokes had never previously witnessed. No aperture was visible from the arterial side, but on the ventricular aspect there was a slit about four lines in length, and one line in width, "through which it was just possible to pass a fine probe." Up to his fatal illness, this gentleman had not exhibited a single symptom of cardiac disease, nor had his medical attendant the slightest suspicion that such existed.

Disease of the aortic valves, as judged by the preceding table (Table X.) is, by a fraction, less common than disease of the mitral valves. It is of more frequent occurrence amongst males than females, in the proportion of 21 : 5. The average age of the male subjects of the disease was forty-two years and eight

* *Opus citat.*, p. 241.

† *Opus citat.*, p. 193.

‡ *The Diseases of the Heart and Aorta*, p. 153.

months; the youngest patient was aged seven, and the oldest seventy years. The average age of the female patients was thirty-two years; the youngest was twenty-six, and the oldest forty-four, years. Calculated in decennial periods, and grouped according to sex, the ages of these twenty-six patients were as follows:

				Males.		Females.
Under 10 years	1	...	0
10 to 20	"	1	...	0
20 to 30	"	3	...	3
30 to 40	"	5	...	1
40 to 50	"	3	...	1
50 to 60	"	2	...	0
60 to 70	"	5	...	0
70 and upwards	1	...	0
				<hr/> 21		<hr/> 5

It thus appears that disease of the aortic valves is essentially a disease of advanced life, and of the male sex. Of the 21 examples amongst males in my list, no less than 14 occurred between the ages of thirty-five and seventy years inclusive, and 10 between forty-five and seventy. In the female, the period of greatest liability is between twenty and fifty years; more than half the entire number of examples having occurred between the ages of twenty and thirty.

Doctor Milner Fothergill is of opinion, that disease of the aortic valves is more serious in the child than in the adult; because in the former it is more rapid in its progress, and usually leads to inadequacy of the valves rather than obstruction at the orifice.* It is, no doubt, in a certain sense more serious in childhood, in consequence of the greater excitement to which the heart is liable, and the more active nutrition incidental to it at this period of life. That regurgitation rather than obstruction is the usual result of disease of the aortic valve in childhood, I agree with Dr. Milner Fothergill in holding. I believe the difference in this respect between the child and the adult is to be explained by the fact, that in the former, valvular changes are due to the incorporation of the products of acute inflammation, or cell-growth; whereas, in the adult and aged subject, they arise

* *Lancet*, May, 1874.

from atrophic changes, by which the valves are retracted and coiled up, and so rendered not only incompetent, but obstructive also.

Disease at the orifice of the aorta, moreover, in the great majority of examples, is unassociated with rheumatism in its origin. Out of 47 cases of valvular lesion reported by Dr. Barclay,* the aortic valves were affected in 28, 13 of which only were reputedly of rheumatic origin; of the latter, the mitral was likewise involved in no less than 12 instances.† Again, out of a total of 113 cases in which he found traces, more or less pronounced, of atheroma in the root of the aorta or in the aortic valves, 15 only yielded a history of rheumatism.‡

Of the foregoing 26 cases of aortic valve-lesion, collected by me, and made the subject of *post mortem* examination, only 2 were associated with a history of rheumatism. In 1, the disease originated with scarlatina, in 5 it was traceable to alcoholism, and in 3, to rupture of the valves by shock. I believe, however, that in these last mentioned cases, as in all similar examples, the valves were in an unhealthy condition; most probably undergoing atheromatous change at the date of the accident by which they were ruptured.

Alcoholism, then, amongst the humble classes, is *par excellence* the cause of disease of the aortic valves. Amongst the rich it is most frequently traceable to gout or the lithic acid diathesis.§ In both classes, and under the circumstances mentioned as peculiar to each, the pathological change by which the valvular lesion is accomplished is initiated by hyperæmia, or inflammatory irritation of the sub-endocardial connective tissue of the valves. Continued or repeated strain of the valves, to whatever cause due, may have a similar effect, and is likely, especially if conjoined with habits of intemperance, or with the gouty diathesis, to issue in disorganization of the aortic valves.

Doctor Clifford Allbutt|| has proved the existence of a special

* *Medico-Chirurgical Transactions*, vol. xxxv.

† *Vide antea*, p. 398. The numbers then given as representing the author's cases of valvular lesion up to that date, now many years ago, have been since greatly augmented.

‡ *Ibid.*, vol. xxxi.

§ *Lithæmia* of Murchison.

|| *St. George's Hospital Reports*, vol. v., and *Clinical Society's Transactions*, vol. vi.

liability to disease of the aorta and its valves, amongst persons engaged in certain avocations, requiring severe, sustained, and oft repeated muscular effort, in which all the muscles of the body are simultaneously engaged, whilst the chest is subjected to compression; for example, amongst "lifters" and soldiers. He has likewise observed similar consequences, in large proportion, amongst smiths and sawyers.

Doctor Peacock declares that he has witnessed them, in many instances, in the persons of young nursemaids, who have been obliged to make great muscular efforts in lifting and carrying heavy children.

The tension to which the aorta is subjected during such efforts, is not unlikely to lead to atheroma of its walls, and aneurism or rupture of the vessel. It is, however, more frequently followed by a slow process of disorganization leading to inadequacy of the valves. Such consequences of severe muscular exertion are not, as Dr. Allbutt shows, common amongst rowers, athletes, and climbers; because, as he believes, the evil conjunction of severe, long continued, and frequently repeated muscular effort, with *compression of the chest*, is not in operation amongst them. There is much force in these reflections, and the conclusions to which they obviously lead should not be lost sight of by Parliament. I should, however, desire to include within the category contemplated by Dr. Allbutt, many classes of operatives not mentioned by him, such as rammers, mill porters, etc., who, though not subjected to thoracic constriction, are obliged to undergo labour too severe and protracted to be compatible with immunity from aortic disease. Compression of the chest during severe muscular effort is not, as far as I know, obligatory upon any body of men except soldiers. These men are not masters of their own actions; they are at the service and the disposal of the state; and to this circumstance, despite the teaching of modern science and the wealth of the country, it is due that, whilst engaged in the physical exertion necessary to qualify them for their important duties, they are forced, and without any conceivable necessity or advantage, to sacrifice their health, and shorten, by several years, the period of their lives. I have already (p. 316) urged this view, on the authority of medical officers of the greatest eminence in the

military service. Dr. Allbutt's conclusions have likewise claimed my attention (p. 485). The eminence of the writer, and the ample opportunities for the special study of this subject which a great manufacturing centre affords, entitle Dr. Allbutt's opinions to the highest respect.

I agree with Dr. Allbutt in the main, and only differ from him as to the extent to which the evils he has so ably exposed are in operation amongst the labouring portion of the community.

The legislature has justly interfered, by the Factory Acts, for the protection of youthful operatives. Why should it not, by a special Act of Parliament, restrict the period of labour of those workmen who are required, by the nature of their avocations, to make extreme and protracted muscular effort?

Atheroma is the usual cause of disease of the aorta and its valves. It was present, as already stated, in 113 out of 419 cases, or in the proportion of 27 per cent. in Dr. Barclay's returns. It is essentially a change of senility, and depends upon nutritive irritation conjoined with inadequate capacity for nutritive change.

Doctor Moxon is of opinion that it "is in continuity with arteritis, and graduates from a condition in which no inflammatory results can be found, into one in which inflammation is unmistakably present."*

Atheroma is most frequently associated with lithæmia, and in many instances, likewise, with a granular or "gouty" condition of the kidneys. Hypertrophy of the left ventricle, to whatever cause due, leads directly to atheroma of the aorta, by subjecting its walls to severe and all but constant tension. The liability will be still greater, if, as constantly happens in gouty subjects, renal disease be likewise present; the walls of the arteries are then inadequately nourished, and arterial tension is increased by the resistance offered to the circulation in the hypertrophied arterioles. But atheroma of the aorta extends ultimately to the aortic valves; and, as Dr. Milner Fothergill has shown,† by impairing the elasticity of the aorta, it becomes a cause of imperfect coronary circulation. Hence, impairment of nutrition, fatty soften-

* *Guy's Hospital Reports*, vol. xvi., third series, p. 431.

† *Lancet*, May 30th, 1874.

ing, and general dilatation of the heart. In the early stage of ventricular hypertrophy, whilst the walls of the ventricle and of the aorta are still structurally sound, the coronary arteries are themselves liable to become atheromatous, from the tension to which they are subjected by the elastic recoil of the aorta; a recoil, be it remembered, which is proportionate to the force of ventricular systole.

Virchow declares that chlorotic females are liable to a special form of disease of the aorta, which may lead to inadequacy and valvular disease. He has found the heart and aorta congenitally undeveloped in the subjects of chlorosis. The aorta was small in calibre, and thin. The changes in the heart were less constant; it was usually small, but in some instances hypertrophied and dilated; differences which, in his opinion, depended upon the state of nutrition, the volume of blood to be circulated, and the obstruction to be encountered in the aorta. Defective development predisposes to disease; hence, diffuse fatty degeneration of the *tunica intima* of the aorta, followed by erosions, most frequently created in the posterior wall of the aorta between the origins of the intercostal arteries.

This change in the arterial coats differs from atheroma, which commences with sclerosis of the deeper coats of the vessel, and less frequently in fatty degeneration of the *tunica media*.*

Fatty degeneration of the heart, from the same cause, is of not unfrequent occurrence in young chlorotic females. Case 29 (p. 550) is a good example of this kind.

Acute inflammation of the aortic valves is followed by thickening, and consequent obstruction or inadequacy, or both these results, in the manner previously described (p. 800).

The changes to which these valves are liable under the influence of chronic irritation, to whatever cause due, may lead to fibro-cartilaginous thickening, or to atheromatous or calcareous transformation. The base of the valve-segments is usually the primary seat of these changes, as remarked by Corvisart; and next in order, the corpora Arantii. From these points the morbid process gradually extends over the entire valve.

Hope remarked that when the central portion of the segment

* Abstract in *London Medical Record*, January 8th and 15th, 1878.

is solidified, the entire valve is subsequently curled up, either athwart the orifice, or against the wall of the artery. In the latter event, inadequacy of the valves, with only trivial obstruction would result, and in the former decided obstruction and incompetency.*

Corrigan classifies the changes to which the aortic valves are liable, under four heads; viz., a reticulated condition; rupture and depression; tightening or curling up against the wall of the vessel; inadequacy of the valves by dilatation of the aorta.†

These lesions may, I think, be reduced to the five heads of

- Thickening;
- Foreshortening;
- Depression;‡
- Perforation;
- Detachment.

Thickening is the result of inflammation of the valves, more or less acute. Foreshortening arises from chronic inflammation leading to abundant cell-proliferation, and subsequent atrophic changes; the valve may be simply retracted, or it may be rolled up, and at the same time elevated against the wall of the vessel, or lowered. Depression arises from over-extension of a thickened and diseased valve. One segment only was affected in the examples which came under my notice; and, except thickening, extension, and displacement by depression, whence resulted extreme incompetency, there was no observable lesion of that segment. Perforation may be the result of rupture or laceration of an unsound valve under high pressure, or of valvular aneurism; it may be likewise caused by the slower process of ulceration, a morbid change of extreme rarity. Partial detachment of one or more of the segments from the root of the vessel may occur under circumstances similar to those which cause laceration. Obstruction, or incompetency of the valves, or both, will be the result of these several changes, according to the actual position and state of the valves.

According to Dr. Peacock, the causes of incompetency of the

* *Opus citat.*, p. 386.

† *Edinburgh Medical and Surgical Journal*, vol. xxxvii., 1832.

‡ I have never witnessed complete retroversion of the valves.

aortic valves may be classified under the following seven heads ; viz., 1. Congenital malformation ; 2. The segments cohering and not growing proportionately to the heart and aorta ; 3. Gradual stretching of the valve, and lowering of its angles of attachment to the vessel, from great and continued muscular effort ; 4. Enlargement of the orifice of the aorta ; 5. Rupture of valves, owing to inadequate support from a dilated left ventricle ; 6. Tunneling beside the valves, in cases of great and long continued obstruction at the orifice of the vessel.*

The *symptoms* of disease at the orifice of the aorta may be conveniently grouped under the two heads of the obstructive and the regurgitative form of valve lesion. With the single exception of the pulse, it may be confidently asserted, that whilst many of these symptoms afford presumptive evidence of either lesion, they all have reference to the consecutive changes of hypertrophy, dilatation, and tissue-softening of the heart. The valve-changes, except in the case of rupture or detachment of the segments, are not directly productive of such inconvenience as to constitute a special and characteristic group of symptoms. By leading, however, to hypertrophy and dilatation of the heart, and fatty degeneration of its structure, they become indirectly the source of symptoms of the gravest augury. Manifestly, therefore, such symptoms, dissociated from physical signs, would possess no specific value as evidence of valvular lesion. Hence, the writings of the many great physicians who preceded Laennec, though full of the most valuable pathological instruction, are, in regard to diagnosis, where not positively erroneous, wanting in the point and clearness of definition which auscultation could alone confer.

Hope has clearly apprehended the distinction between the symptoms due to the valve-lesion, and the graver indications of consecutive changes in the cavities and walls of the heart. He has, however, mistaken hypertrophy for one of the conditions upon which the symptoms connected with the advanced stages of valvular disease depend. Until softening and dilatation ensue, hypertrophy, being itself a condition of compensation, will cause no greater inconvenience to the patient than that of

* *St. Thomas's Hospital Reports*, new series, vol. ii., 1871, p. 234.

strong pulsation after exercise or excitement. It is, however, of evil augury, as a preliminary step towards the series of retrograde changes comprised in softening and dilatation. In cases of simple obstruction at the aortic orifice, the pulse, as truly remarked by Hope, exhibits no special character, unless the contraction of the outlet be extreme. In the latter contingency, the pulse will be weak; but, owing to the usual accompaniment of atheromatous change in the coats of the arteries, it will retain a certain degree of apparent fulness and volume. Irregularity and intermittence of the pulse belong, essentially, to the consecutive changes of softening and dilatation of the left ventricle, and therefore may or may not be present.

Corvisart held that the pulse is permanently irregular in all such cases; but, his statement has reference to the advanced stages, where softening and dilatation are well established.

Where inadequacy of the valves exists, however, the pulse is in the highest degree characteristic. It was described by Hope as a "jerking" pulse, or a pulse of "unfilled arteries," and by Sir Dominic Corrigan as a "vibrating" pulse. To the latter author belongs the great merit of having laid down rules by which the lesion may be identified, and of having been the first to present the entire subject in a definite scientific aspect.

In the first edition of his standard work *On Diseases of the Heart and Great Vessels*, in 1831, and therefore a year before the publication of Corrigan's paper, Hope correctly described the pulse of aortic regurgitation; but he erroneously ascribed its distinctive peculiarities to accompanying carditis, or adhesion of the pericardium. He soon, however, corrected this error, on finding the "jerking" pulse actually present where aortic regurgitation alone existed, but not before Corrigan's paper had appeared (1832). Both authors are, therefore, entitled to the merit of originality in their respective publications.

Hope was of opinion that the throbbing pulse of aortic reflux differed from that of anæmia, in being at once abrupt, and repeated at distant intervals, *celer et infrequens*. In none of Corrigan's cases was the pulse under 80 in the minute. In aortic patency, unaccompanied by obstruction, I have invariably found the pulse infrequent.

The designation of "jerking," and "collapsing," are strictly applicable to the pulse of aortic regurgitation; the former implying that, from a state of emptiness, the artery is suddenly filled with blood; and the latter, that, from a state of repletion, it is suddenly emptied. It has been likewise described as the "water hammer," and the "splashing" pulse; and, after the distinguished author of the memoir on *Permanent Patency*, "Corrigan's pulse." This pulse is characterized by suddenness of shock, rapidity and completeness of collapse, magnitude of volume, and infrequency of repetition. The abruptness of the pulse, as recognized by touch, and as shown by the verticality of the up-stroke in the sphygmographic tracings, is due to the sudden distension of a previously empty vessel. The rapid collapse of the artery depends upon the sudden emptying of the aorta through the inadequate valves, at the first moment of ventricular diastole.

M. Aran has remarked that in the progress of the disease, the pulse ceases to exhibit the "collapsing" character, owing to the circumstance, that at this period the coats of the arteries have become rigid by atheromatous change.*

The magnitude of the pulse is accounted for by the previous dilatation of the artery, and the injection of a large volume of blood from an hypertrophied and dilated ventricle. The infrequency of pulsation is proportionate to the dilatation of the ventricle, and the volume of blood circulated at each systole.

Doctor Stokes has described,† under the designation of "steel hammer" pulse, a peculiar and characteristic pulsation of the arteries which may be witnessed in cases of acute rheumatic arthritis supervening upon chronic inadequacy of the aortic valves. The pulse is abrupt and energetic, as the rebound of a smith's hammer from the anvil; it is exhibited, however, only in the arteries adjacent to the affected joints.

Fig. LI. (p. 870) illustrates the graphic character of the pulse of simple aortic obstruction. The five subjoined tracings (Figs. XLVI. to L. inclusive) are characteristic of the pulse of aortic patency. The three latter are borrowed, by kind permission of the author, from Dr. Boileau's excellent little work.‡

* *Archives Générales de Médecine*, tom. xv., 1842.

† *Continued Fever*, 1874, p. 244.

‡ *The Sphygmograph*, by J. P. H. Boileau, A.B., M.D., 1874.

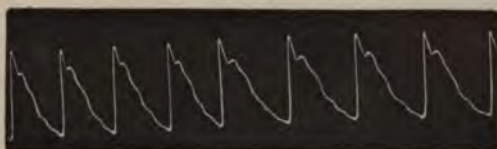


FIG. XLVI.

Aortic insufficiency. (E. G.)

This tracing indicates hypertrophy and dilatation of the left ventricle, with inadequacy of the aortic valves. Its distinctive features are, great height and verticality of up-stroke, pointed summit, well-marked tidal, and absence of dicrotic wave. Pulse regular.

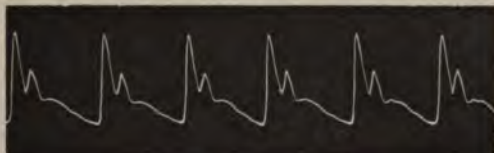


FIG. XLVII.

Aortic insufficiency. (G.)

From Dr. Grimshaw. Conditions indicated same as those in Fig. XLVI, especially the length and vertical ascent of the percussion-stroke, and the height of the tidal wave; the former indicating great force of ventricular systole, and the latter considerable dilatation of the left ventricle.

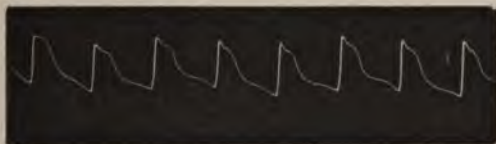


FIG. XLVIII.

Aortic insufficiency; cardiac hypertrophy (33 ozs.); adherent pericardium; ventricular dilatation. (Dr. Boileau, Fig. VII.) Pulse 110. Respiration 46.

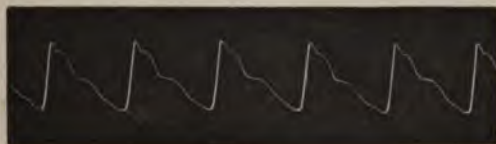


FIG. XLIX.

Aortic insufficiency. (Dr. Boileau, Fig. XXXI.)



FIG. L.

Aortic insufficiency. (Dr. Boileau, Fig. XXXII.)

Doctor Henderson maintained that the pulse is further characterized by postponement, in relation to the cardiac impulse and first sound.* According to him, the radial pulse alternates with the impulse of the heart, "or occurs in the middle of the interval between the successive pulses of the heart." I cannot confirm this observation; indeed I am convinced it is erroneous. The arterial pulse depends, as to rhythm, upon the ventricular, not upon the aortic, systole. The characters of the pulse-tracing show that the radial pulse is in normal relationship to the ventricular systole.

A pulse possessing these characters, is, in the strictest sense, pathognomonic. It indicates, however, extreme inadequacy of the aortic valve, and is not present when a mere leakage exists at the orifice. In the early stages of the pathological change by which incompetency of the valve is produced, it must not, therefore, be looked for; and, as a guide to treatment, it is less valuable than diastolic murmur, because developed at a later period of the disease. It may, however, be a primary manifestation of valvular inadequacy, and coeval with murmur. Such is the case when it is suddenly produced by rupture, laceration, or retroversion of the valve. If, during a shock or strain, a sensation is experienced of something having given way within the chest, accompanied by sharp precordial pain, palpitation, dyspnoea with or without hæmoptysis, oppression, and faintness; and if, at the same time, right basic diastolic murmur be suddenly developed, the diagnosis of rupture, detachment, or retroversion of one or more segments of the aortic sigmoid valve may be confidently made. Fulness of volume, though usual, is by no means a constant characteristic of the pulse of aortic regurgitation;

* *Edinburgh Medical and Surgical Journal*, vol. xlviii., 1837.

where obstruction likewise exists, it will be small and flickering. On the other hand, as already stated (p. 810), it may present a factitious fulness, even when obstruction coexists, owing to atheromatous rigidity or calcification of the arteries.

Corrigan was the first to direct attention to the remarkable symptom of "visible pulsation" of the arteries of the head, neck, and upper limbs, as characteristic of aortic patency. The throbbing of the radial and ulnar arteries is strikingly exaggerated by raising the arm above the head; it is likewise exhibited in the temporal arteries; but in minor degrees of inadequacy it does not exist in these vessels, and, when present, it is always less pronounced than in the arteries of the wrist. Visible pulsation of the arteries is not, however, pathognomonic of aortic patency; it may exist under two conditions in the absence of inadequacy of the valves; namely, when the arteries have become rigid and tortuous by atheromatous change, and when copious hæmorrhage has taken place. In the former of these conditions, visible pulsation of the superficial arteries is permanent; they are lifted visibly from their bed at each systole of the left ventricle, as the result of the elongation and straightening of the vessels, produced by their sudden distension with blood. The pulse, in such cases, would be most appropriately designated as "locomotive." In the latter condition, visible pulsation is only temporary, and coeval with insufficiency in the *volume* of the blood; but it is, nevertheless, a veritable pulse of "unfilled arteries." The differential diagnosis is, however, not difficult. Where the arteries pulsate visibly by reason of the rigid state of their walls, this condition is readily detected by the finger; it is, moreover, unassociated with sudden collapse of the vessel, which is manifestly tortuous, and the patient is also most frequently of advanced age. The principal graphic features of such a pulse (*pouls des vieillards*) as exhibited in a sphygmographic tracing; namely, great amplitude, vertical upstroke, and an extended summit, contrast strikingly with those of aortic patency. Fig. LI. illustrates these features, and may be compared with Figs. XLVI. to L. inclusive.

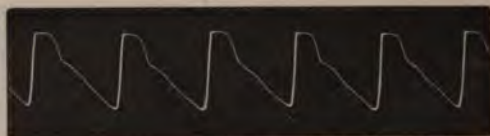


FIG. LI.

The pulse of advanced age. (Dr. Boileau, Fig. XVIII.) From a military officer, aged eighty, who had fought at Waterloo. He was in vigorous health when this tracing was taken.

Throbbing from loss of blood is associated with recent hæmorrhage. In all cases, however, the crucial test of auscultation is necessary to establish the diagnosis.

The breathing may be scarcely quickened in the state of repose, but it is readily accelerated by even moderate exercise, and is then accompanied by palpitation. Corrigan truly remarks that hæmoptysis is rare, and that œdema, when present, is always a late symptom. When, however, in the progress of the disease, the mitral valve has become inadequate by thickening of its proper structure, consequent upon the extreme and protracted tension to which it has been subjected, and by dilatation of the ventricle, pulmonary congestion and hæmoptysis, with general venous engorgement and dropsy, may ensue. But death by exhaustion, from failure of the left ventricle, usually anticipates these consequences, except when inadequacy is suddenly produced by rupture or detachment of the valve. In such contingency the left ventricle is surprised in a state of comparative unpreparedness; hence, syncope, engorgement of the lungs, and most frequently hæmoptysis, great respiratory distress, general venous stasis, cyanosis, and partial or complete failure of the radial pulse. Death is most frequently the result of such an accident in young subjects, within a few months of its occurrence, because the valves, the kidneys, and the aorta, being in such cases usually unaffected by previous disease, the left ventricle has not undergone the preparatory change of hypertrophy. But in the adult, the ventricle is most frequently in a state of partial hypertrophy when rupture of the valve occurs, owing to the previous existence of valvular, renal, or aortic disease; or perhaps of all these combined. In the three examples of this accident recorded by

Dr. Balthazer Foster,* death followed within three months, eighteen months, and twenty-three months of the date of its occurrence, respectively. He adds, that in no well authenticated case has the occurrence of this accident been referred to a period more remote than four and a-half years previous to the death of the patient.

Four examples of rupture of the aortic valve, verified by autopsy, have come under my notice. The date of the accident was fixed with approximate accuracy by the attendant circumstances in all four cases.†

In one case, that of a woman, aged forty-four, death took place on the next day after the accident. The heart was fatty, the aorta was dilated, and the aortic valves were calcified. In the remaining three cases the patients were men, aged, respectively, thirty, thirty-four, and forty-five years; death followed the accident of rupture of the valve at intervals of two years, in the case of the two former, and nine weeks in that of the latter.

Death may follow the occurrence of rupture at a very early period also, in cases of advanced fatty degeneration of the left ventricle, as exemplified in Case 91, p. 873. In such, the ventricle is overwhelmed by the shock of the accident; being structurally unsound, it is unable to recover its equilibrium, and, owing to impairment of nutrition, it is incapable of undergoing changes of compensation.

Dilatation of the root of the aorta, by divaricating the valve-segments, deprives them of the support which, in the state of health, they afford one another by the mutual and close application of their *lunulæ*, and thus becomes a predisposing cause of rupture.

Amongst the accidental symptoms due to intercurrent congestion of the viscera are, oppression, starting from sleep, orthopnoea, angina, cedema of the lower extremities and genitals, and enlargement with tenderness of the liver. Congestion of the kidneys is indicated by albumen in the urine, but without reduction in its specific gravity, except where chronic renal disease likewise exists.

The *physical signs* are eminently characteristic, and are com-

* *Clinical Medicine*, 1874, p. 189.

† See Cases 25, 91, 98, and 99.

prised under the three heads of, impulse, area of percussion-dulness, and murmur.

In the stages of disease at the aortic orifice, both obstructive and regurgitative, previous to retrogressive change of the substance of the heart, the cardiac impulse is strong; and in those rare cases of simple obstruction in which retrogressive tissue changes in the heart are not coeval with lesion of the valves, it is remarkably so, contrasting with the radial pulse, which is small and feeble; but subsequently to the occurrence of fatty metamorphosis the impulse is feeble; and in the later stages of the disease it is irregular, unequal, and occasionally intermittent.

In simple obstruction the point of apex-pulsation is displaced outwards, proportionately to the degree of hypertrophy of the left ventricle; in such cases, however, it rarely transgresses the nipple line by more than half an inch. But where valvular inadequacy, with or without obstruction, has been long in operation, the outward displacement of the apex is much greater, owing to the coexistence of dilatation of the ventricle. In extreme degrees of inadequacy of the aortic valve the right ventricle is also dilated, and in such cases I have found the apex-pulsation two and a-half inches to the left of the nipple line. Owing to the continued action of the reflux current upon the apex of the ventricle, that chamber is likewise elongated; hence, the point of apex-pulsation is displaced downwards, and usually occupies the sixth intercostal space; it may be detected beneath the seventh rib, and, in extreme cases, even in the seventh intercostal space.

The area of percussion-dulness is proportionate to the enlargement of the heart; but where pulmonary emphysema likewise exists, the area of dulness is proportionately restricted. The dulness extends horizontally rather than vertically; and, owing to the persistence of thoracic vibration, it yields a sensation of only partial resistance; in both these respects contrasting with pericardial effusion as previously described.* It is further distinguishable from that due to pericardial effusion, by the circumstance that impulse is more or less distinctly perceptible over the entire area of dulness.

* See p. 388.

Murmur is, however, the only pathognomonic sign of disease of the aortic valves. Alone, it suffices to warrant a positive diagnosis; and without it, all other signs are only presumptive. The diagnostic value of aortic murmur depends upon a correct appreciation of its rhythm, seat, and transmission. For the purpose of differential diagnosis, it is further necessary to bear in mind that the murmur may be single or double, according to the actual condition of the valves; the former indicating obstruction *or* regurgitation, as determined by the rhythm; and the latter, usually designated the "see-saw" or "to and fro" murmur, obstruction *and* regurgitation. Owing to the want of a precise distinction as to these details, the memoir of Corrigan, to whom medical science is indebted for the first complete exposition of this subject, is in some degree vitiated; but the period at which he wrote may be held as sufficient excuse for this partial defect; and the great merits of his article on "permanent patency," are scarcely diminished by it. It is necessary, however, in a modern treatise on the subject, to point out where, and in what, he has fallen into error.

Corrigan recognized only two causes of murmur in the aorta; namely, inadequacy of the valves, and aneurism. Hence have arisen certain cardinal errors in his identification of the rhythm of aortic murmurs, and in his diagnosis of the precise condition of the aortic valves. He ignored the murmur arising from obstruction at the orifice of the aorta; and when this occurred singly he regarded it as diastolic in rhythm, and as evidence, not of obstruction, but of inadequacy of the valves. The murmur is audible, according to him, in the carotid and subclavian arteries; it accompanies the "visible pulsation and diastole of these vessels," and the *frémissement* perceptible in them. A murmur answering to this description would manifestly be systolic in rhythm, and, having its seat at the orifice of the aorta, it would indicate obstruction of the valves. When two murmurs are audible in the aorta, he would regard both as arising from "great deficiency of the valves:" the first, as "from a rushing of blood up the aorta, the second from a rushing of it back into the ventricle."

The murmur indicative of obstruction at the aortic orifice is

strictly systolic in rhythm; it more frequently accompanies than replaces the first sound of the heart. When, however, the mitral and tricuspid valves are thickened or incompetent; also when the myocardium is in an advanced stage of fatty degeneration, the murmur of aortic obstruction will entirely replace the first sound.

Flint says it is oftener soft than harsh. I cannot agree in this opinion. Indeed, I have never heard a veritable aortic systolic murmur which was soft in quality; harshness is one of its proper, if not inseparable, characteristics. It is *always* transmitted into the aorta and the great arteries of the neck; but it is certainly not louder, as Flint maintains, in the right second intercostal space than at midsternum; this latter situation is, in my judgment, its point of maximum intensity. It is usually audible at the apex of the heart; and if very loud at the base, it may be heard likewise in the thoracic and abdominal aorta; but in such cases it is reinforced in the aorta by a rough and atheromatous condition of the walls of that vessel. The line of upward transmission coincides with that of the arch of the aorta, and therefore deviates somewhat to the *right* of the mesial line. Exceptions to this rule are extremely rare. Dr. Hawtrey Benson, however, has recorded one such example.* In this case, a systolic aortic murmur was transmitted upwards and to the *left*; the arch of the aorta was found, after death, to take a similar course. When audible in the back, which is exceptional, save when it is transmitted into the descending aorta, the murmur of aortic obstruction is located in the left interscapular space. As Stokes avers, murmur of this rhythm and seat is usually associated with fatty degeneration of the heart; and, I will add, with an atheromatous condition of the aorta and principal arteries.

It may be confounded with hæmic murmur at the base of the heart; with the murmur of aneurism of the ascending portion of the arch; and with that produced by a rough and scabrous condition of the first portion of the aorta.

From hæmic murmur it may be distinguished by its harsh quality, by its being audible in the carotid and subclavian arteries in the *recumbent posture*, by the age and sex of the patient,

* *Proceedings of Pathological Society of Dublin*, new series, vol. iv., part i., p. 34.

and, frequently, by the coexistence of atheroma of the superficial arteries. The patient is usually of middle or advanced age, and of the male sex; whereas, the subjects of hæmic murmur, not consequent upon recent hæmorrhage, are in almost all instances young females. When the murmur is purely hæmic, it is not audible in the cervical arteries in the recumbent posture, unless there be also a general arterial, or a localized subclavian murmur; and further, it is associated, in almost every instance, with the characteristic hum in the jugular veins.

The murmur of aneurism is usually localized at a higher point than the base of the heart, and is, moreover, characterized by one or more of the other signs proper to that disease. Aneurism may, however, exist at the root of the aorta, and give rise to inadequacy of the valves through dilatation of the vessel, as Corrigan showed; or it may be associated with valvular obstruction and inadequacy. Of the latter combination, a good example has been published by Dr. McAdam.*

Finally, murmur produced by a scabrous condition of the ascending aorta, in the absence of valvular lesion, an exceedingly rare phenomenon, of which I have met with only one example (Case 62, p. 713), will be characterized by a somewhat higher seat of origin than the base of the heart, and by its *not* being transmitted into the carotid arteries. Elsewhere† I have ventured to suggest that the absence of arterial murmur in such cases may be due to the superficial, as distinguished from the axial and ordinary seat of origin of the phenomenon. It is, moreover, loudest in the curvature of the arch, or at the right second costal cartilage.

Sir Dominic Corrigan exhibited before the Pathological Society of Dublin‡ the heart of a young man, in which the root of the aorta had undergone complete osteoid transformation; it was likewise greatly dilated, and the aortic valves had been rendered thereby inadequate. During the patient's last illness, a systolic murmur of metallic quality, appropriately designated a "trumpet-bruit," was audible at the base, and in the ascending aorta and

* *Dublin Journal of Medical Science*, vol. ix., 1836.

† *Proceedings of the Pathological Society of Dublin*, vol. vii., p. ii., new ser., p. 139.

‡ See *Proceedings*, vol. ii., p. ii., new series, Feb. 1864.

carotid arteries; there was likewise a soft diastolic murmur. Sir Dominic Corrigan refers to a similar specimen which he had previously exhibited, and to another which had been brought under the notice of the Society by Dr. Banks. In this latter case, however, there had been a double "trumpet-bruit," which was found to have been caused by a "tongue of bone" projecting into the orifice of the aorta. He regards a "trumpet-bruit" as absolutely diagnostic of bony deposit in the aorta, either in the form of a "rim of bone," or a "projection or tongue of bone."

But, independently of all these causes, and in the absence of disease of the aorta or its valves, and of anæmia, systolic murmur in the aorta, propagated into the carotid and subclavian arteries, may be produced by disease of, or deposit upon, the anterior segment of the mitral valve, as exemplified in Case 31, p. 554. This rare form of lesion is most likely to be confounded with obstructive disease at the orifice of the aorta; and in the present state of science, the differential diagnosis, as between the two conditions, is, in practice, quite impossible. Theoretically, it may be supposed that, where the murmur is caused by mitral lesion, its point of maximum loudness would be somewhat below and to the left of the aortic orifice; but this is a mere hypothesis, devoid of clinical basis.

Cardiac fremitus, transmitted into the arteries of the neck, is an occasional, but by no means frequent concomitant of systolic aortic murmur of valvular origin. Fremitus may likewise attend a vascular aortic murmur; but in this association it would not, according to my observation, be perceptible in the carotid and subclavian arteries. Fremitus indicates either an extremely irregular, and, most frequently, a spiculated state of surface at the seat of its origin, or a pendulous or vibrating flap of fibrin, or of lacerated valve. I have not met with a single example of it in association with the murmur of aortic reflux, and I incline to believe that it is never so associated.

Fremitus is, therefore, intimately connected with systolic aortic murmur in origin, in rhythm, and in transmission; when it is present, murmur may be also confidently sought for; but it may be absent where murmur exists, because it requires for its production a more irregular state of surface, and more intense vibration than does the latter phenomenon.

Both the murmur of aortic obstruction, and the attendant fremitus, may be temporarily or definitively suspended by extreme debility of the left ventricle; but, so far as I have been able to observe, this occurs only at a period of the disease when there is other and indubitable evidence to show that death is close at hand.

The murmur of aortic regurgitation is most frequently diastolic in rhythm; that is to say, it occupies the period of the second sound. It may be substitutive, or accompanying; usually the latter, because the pulmonic second sound is in most cases audible in some degree at the right base. In quality it is, according to my experience, almost invariably soft and blowing, but it is occasionally attended by a musical note. It is loudest at mid-sternum, and transmitted with greatest intensity in the direction of the right apex. Dr. Foster is of opinion, that the conduction of an aortic diastolic murmur to the apex indicates regurgitation by incompetency of the *posterior* segment of the valve; whereas, conduction to the ensiform cartilage is diagnostic of inadequacy of either or both of the anterior segments.* He adduces two most interesting cases observed by himself, in support of the opinion just stated. They are certainly of a character to lend it support; but more extended observation would be necessary to establish so important a point of differential diagnosis.

In two cases (Cases 25 and 98) I find my notes to bear in some measure upon this question. In the latter (Case 98, p. 884), the murmur, which was aortic in origin and prediastolic in rhythm, was heard at the apex of the heart, and was, owing to that circumstance, at first regarded as presystolic. On *post mortem* examination the right anterior segment was found ruptured. In the former (Case 25, p. 538), the murmur was diffused universally over the chest; and, on examination of the body, the right anterior segment was likewise found to have been ruptured.

This question has a practical bearing. According to Dr. Foster, inadequacy of the anterior segments is of more serious import, as indicating a shorter period of survival, owing to their relationship to the coronary arteries. My attention has been too recently directed to this point, and my observations have been

* *Clinical Medicine*, 1874, p. 127.

too few, to warrant me in making any definite statement of opinion in relation to it. The murmur is audible, but less distinctly, in the ascending aorta, less constantly in the transverse portion of the arch, and in only a single instance (Case 25) have I heard it even faintly in the arteries of the neck. In that case the aortic valve had been ruptured, and the diastolic murmur was remarkably loud, and diffused over the entire chest. Flint asserts that this murmur is loudest in the third or fourth left intercostal space, close to the sternum. I cannot subscribe this statement.

I quite agree with Walshe in regarding the association of tactile fremitus with the murmur of aortic regurgitation, as exceedingly rare. This author declares he has met with only a single example of it.* Sir Thomas Watson has published another.† No example of the kind has come under my observation. Considerable discrepancy of opinion exists as to the transmission of aortic diastolic murmur into the carotid and subclavian arteries. I have already stated that, in my opinion, this is in the highest degree exceptional. In reference to this point, however, I find myself at issue with very eminent authority. Sir D. Corrigan, as already mentioned, regarded carotid and subclavian murmur as one of the distinctive signs of aortic regurgitation; but I have already shown that the cases from which he drew this conclusion were, most probably, examples of the double lesion. M. Guizot, who wrote shortly after the date of Corrigan's memoir, likewise regarded carotid and subclavian murmur as a concomitant sign of aortic reflux;‡ and M. Charcelay, two years later, expressed a similar opinion.§ Flint holds|| that this murmur is "frequently but not invariably" heard over the carotids. Professor Gairdner declares that aortic diastolic murmur is propagated, "though sometimes very faintly," into the arteries of the neck.¶ M. Aran asserts that it is transmitted not only into the cervical arteries, but likewise into the brachial, crural, and tem-

* *Diseases of the Heart*, fourth edition, 1873, p. 34.

† *London Surgical Gazette*, vol. xix.

‡ *Archives Générales de Médecine*, tom. v., 2eme série, 1834.

§ *Ibid.*, Mars, 1837.

|| *Diseases of the Heart*, second edition, 1870, p. 219.

¶ *Clinical Medicine*, 1862, p. 587.

poral arteries.* Dr. Foster has heard this murmur in the carotid arteries, as I have at least in one instance, but he does not assert that it is usually audible† in these vessels. Independently of the teaching of actual clinical experience, it is scarcely conceivable, on theoretic grounds, that a murmur of low pitch, as that of aortic patency certainly is, should be transmitted to a point so distant, *against* the current by which it is produced.

Murmur of aortic patency may, however, be *prediastolic* or *postdiastolic* in rhythm. Examples of both these modifications of diastolic murmur in the aorta will be found amongst the cases appended to this section. I believe both are produced by leakage at the orifice, arising from defect of the valves, but in different degrees. One or two segments of the valve, or the principal portions of all three, are structurally healthy, and capable of yielding a clear second sound; but in some instances this sound is *preceded* by a faint murmur of reflux, arising from impediment to the free and rapid closure of the valves (*prediastolic*). The retardation of closure may be caused by adhesion of the segments to one another; by attachment of one of them to the wall of the aorta by means of a fibrous band; or, as in Case 98, the murmur may result from perforation and permanent depression of one of the valve-segments.

In other instances, a second sound, more or less normal in character, is immediately *succeeded* by murmur (*postdiastolic*). This may be produced either by a regurgitant axial current from retroversion of the edge of one of the valves, or from thickening and maladaptation of the segments, at the acme of aortic systole. But, *postdiastolic* aortic murmur may be likewise an indirect consequence of reduplicated second sound, owing to dilatation of the left ventricle; the aortic element of the reduplicated sound being posterior in time to the pulmonic, and subsequently being replaced by a murmur, the result of accidental rupture of the valve. Case 99, p. 886, affords a good example of this kind.

Manifestly, a similar result may follow from a slow process of disorganization, leading to incompetency of the valve, provided the left ventricle had been previously dilated, whether

* *Archives Générales de Médecine*, tom. xv., 1842.

† *Clinical Medicine*, 1874, p. 127.

from renal disease, or atheroma of the aorta, followed by softening of the heart. Case 28 supplies an illustrative example.

I cannot agree with Flint in the opinion, that prediastolic murmur may be due to atheroma of the aorta. Diastolic aortic murmur is not necessarily permanent, as Walshe and Sanders* allege; it may be suspended from two different causes; namely, temporary restoration of valvular competency by the entanglement of a plug of fibrin in the abnormal passage, and debility of the left ventricle from fatty degeneration, combined with a rigid and inelastic state of the aorta. Dr. Gairdner has published an example of the former kind,† and I have met with instances of the latter.

Diastolic basic murmur is, in almost every instance, due to inadequacy of the aortic valves. It may, however, arise also from regurgitation in the pulmonary artery; aneurism of the aorta immediately above the valves; patency of the ductus arteriosus, and aortic aneurism communicating with the pulmonary artery. Inadequacy of the pulmonic semilunar valves is the rarest of all valvular lesions; a murmur due to this cause would be loudest in the second intercostal space immediately to the left of the sternum, and further, it would be transmitted only downwards and towards the right apex. Aneurism springing from one of the sinuses of Valsalva is usually associated with inadequacy of the aortic valves, and is most likely to be indicated by some of the signs proper to aneurism, especially that of a *booming* quality of the attendant murmur; the latter is, moreover, systolic in rhythm, except in the very few instances in which the mouth of the aneurism is covered by the valve.

Patency of the ductus arteriosus, of which Dr. Hilton Fagge has recorded an example,‡ is indicated by murmur located considerably to the left of the sternum, and faintly transmitted downwards and to the right. Aneurism opening into the pulmonary artery is most frequently attended by murmur of systolic rhythm; and even when the murmur is diastolic, its seat of maximum intensity and line of transmission would probably suffice for the purpose of differential diagnosis.

* *Edinburgh Medical Journal*, January, 1868.

† *British Medical Journal*, March 30th, 1872.

‡ *Guy's Hospital Reports*, 1873.

Dr. Bellingham avers that a roughened, rigid, and dilated condition of the ascending aorta, is of itself competent to produce a double murmur, which may be readily mistaken for that arising from lesion of the valves.* The systolic murmur depends upon the rough and dilated state of the vessel; the diastolic murmur, in such a case, he would explain on the assumption, that whereas, during ventricular systole, the dilated portion of the vessel is distended with blood, during diastole it is not distended, but presents, owing to its want of resiliency, a virtual vacuum, into which the blood rushes from the carotid and subclavian arteries, under the reaction of their healthy walls. This explanation he would likewise apply to the double murmur of aneurism in this situation. But, unfortunately for this theory, *diastolic* murmur in the ascending portion of the arch of the aorta, whether in connexion with simple or aneurismal dilatation of the vessel, where the valves are not thereby rendered inadequate, and are not themselves diseased, is one of the rarest physical phenomena; whereas, both the conditions mentioned as competent to produce it are exceedingly common. I have never met with an example of diastolic murmur in a simply dilated aorta, in which inadequacy of the valves did not likewise manifestly exist. Professor Law, however, has communicated to the Pathological Society† the particulars of a case, accompanied by the morbid specimen, which he regarded as confirmatory of Dr. Bellingham's doctrine.

Jaccoud has endeavoured to show that there is a causal relationship between aortic patency and fatty degeneration of the heart, owing to the inadequacy of volume and the defective pressure of blood in the coronary arteries, which are assumed to be the necessary consequences of the former condition. There may be some ground for this statement; but, it is certain that hypertrophy is an antecedent condition to fatty change; and that fatty degeneration of the heart is no less frequently met with as an ulterior stage of hypertrophy from other causes, and notably, from simple obstruction of the aortic valves. Indeed, Dr. Stokes is of opinion, and I quite agree with him, that simple

* *A Treatise on Diseases of the Heart*, 1857, p. 152.

† Vol. iii., p. iii., March, 1868.

aortic obstruction is all but invariably associated with fatty degeneration of the heart.

Patency of the aortic valves has been regarded as, *par excellence*, the form of organic disease of the heart in which sudden death is to be apprehended; and Walshe, basing his opinion upon eight cases which have come under his notice, goes the length of asserting that the less complex this disease, the greater is the danger of sudden death.* Corrigan, on the other hand maintains, that in simple aortic patency death is never sudden; and under proper restriction and treatment, the patient is not only capable of leading an active life for many years, but is actually benefited by moderate exercise and labour.† He has recorded the following cases in support of this statement: A labourer, aged twenty-four, had been the subject of aortic insufficiency for seven years, during which he continued to work; this man ultimately died of acute secondary endocarditis, but not suddenly. A dispensary medical officer had laboured under aortic patency for fifteen years, and ultimately died of another affection.‡ A young man had been the subject of this disease for a period of eight years, during which he had performed with efficiency the laborious duties of a lamp-lighter, and ultimately died after a protracted illness.§ Dr. Gordon has published the following example of this kind: The patient had been for fifteen years the subject of inadequacy of the aortic valves; during the whole of this period he had worked as a mechanic, without experiencing much, if any, inconvenience from his disease; and death, which took place whilst the man was in hospital, and under Dr. Gordon's treatment, was not sudden.||

I have observed several cases of the same kind, though less striking in character; and I have arrived at the conclusion that, under ordinary circumstances, sudden death is by no means characteristic of disease of the aortic valves, obstructive or regurgitative, or obstructive and regurgitative combined.

When, with disease of the aortic valves, the case has termi-

* *Diseases of the Heart*, fourth edition, 1873, p. 394.

† *Proceedings of the Pathological Society of Dublin*, vol. ii., part ii., new series, 1864.

‡ *Edinburgh Medical and Surgical Journal*, April, 1832.

§ *Ibid.*, March 5th, 1857.

|| *Ibid.*, vol. iii., part iii., new series, p. 276.

nated by sudden death, I believe the usual concomitant or consecutive changes of fatty degeneration of the left ventricle, and dilatation with atheroma of the aorta, have taken place ; and that these changes are mainly, if not exclusively, chargeable with the sudden death of the patient. Case 101, p. 889, may be adduced as a good example in support of this opinion.

The *treatment* of disease of the aortic valves, whether obstructive or regurgitative, should be mainly dietetic and regimenal ; having for its object the postponement of tissue degeneration of the heart and aorta, rather than the amelioration of the actual condition of the valves ; an object which is quite unattainable by any means at present known to medical science. The patient should avoid the use of alcoholic stimulants and malt drinks, beyond a very moderate daily allowance ; also fatiguing exercise or labour, as far as is practicable, especially such as involves raising the arms above the head, or so called "overhand" work ; and, whilst eschewing copious draughts of any kind of liquid, which act injuriously by increasing for the time the volume of the blood, should take plenty of nitrogenous food, of such kind as may best agree with the stomach. Animal fats, sugar, and saccharine vegetables, should be used with great moderation. The bowels should be moved at least once daily, and the skin made to act by gentle exercise, and the occasional use of the warm bath. Vegetable tonics with alkalies, such as infusion of gentian with bicarbonate of soda ; or infusion of calumba or of chiretta, with small doses of carbonate or citrate of lithia, must be administered whenever the stomach and liver, by loaded tongue and lithate deposit in the urine, give evidence of derangement. The body should be warmly covered with the view of maintaining a full and free cutaneous circulation, which acts as a natural derivative from the central organs ; and, for the opposite reason, prolonged exposure to cold should be avoided. In the warm season, however, cold bathing need not be prohibited, provided only the body be not chilled by protracted immersion. A period of a few seconds in the cold water will be quite sufficient ; and the body, whilst in it, should be briskly exercised, and afterwards rapidly dried. Sea-bathing is preferable to any other, because of its stimulant action upon the skin.

If the heart exhibit any signs of weakness, whether in the obstructive or the regurgitative form of lesion of the aortic valve, digitalis with iron, or quinine with strychnia, should be given; the former, when the action of the heart is likewise frequent and irregular, and the latter, where cardiac debility, with vertigo and imminent syncope, are the predominant characteristics. I usually prescribe the tincture of digitalis, in combination with the tincture of the perchloride of iron and spirit of nitrous ether, of each ten minims, to be taken with an ounce of infusion of quassia or of calumba three times daily, continuing the use of this medicine till the pulse-rate has been reduced, and the action of the heart has exhibited greater strength and regularity.*

In 1832, Sir Dominic Corrigan deprecated the use of digitalis in the treatment of aortic patency, on the ground that, by slowing the action of the heart, and thereby protracting the period of diastole, it favoured the process of dilatation of the left ventricle. In 1871,† Dr. Balthazar Foster objected to it for reasons identical with those urged by Sir Dominic Corrigan. He admits, however, that digitalis may act beneficially in this disease, under circumstances indicative of over-compensation; viz., where the heart acts with violence and rapidity, and the arteries are in a state of high tension. In such a case I would prefer the application of a few leeches at midsternum, and subsequently, ten-drop doses of the tincture of aconite (B. P.), given every third hour till cardiac excitement had subsided. The tendency to syncope, which Dr. Foster and others have witnessed under the use of digitalis in this and other forms of organic disease of the heart, is, I incline to think, occasionally due, not to the drug itself, but to the mode of its administration. I believe the infusion is the most objectionable form in which it can be given, having observed unpleasant consequences, similar to those mentioned by Dr. Foster, resulting from it. But, given as the tincture, and combined with iron and ether as previously suggested, I have rarely known digitalis to cause un-

* *Vide* p. 667.

† *British and Foreign Medico-Chirurgical Review*, No. xcv.; see also *Clinical Medicine*, 1874, p. 107.

pleasant symptoms; on the contrary, in numerous instances it has been followed by the most favourable and satisfactory results. I have, in a few instances, been under the necessity of suspending the use of digitalis owing to threatened syncope; but in the cases referred to there was fatty degeneration of the heart, in some instances with, and in others without, disease of the aortic valves. Whilst admitting that infrequency of cardiac action does not, of itself, constitute an indication for the use of digitalis, I cannot regard it in the opposite light, as prohibitory of that agent. Mere infrequency of pulse does not indicate prolongation of aortic recoil, and of influx into the left ventricle, it only shows that the rhythm of the heart is slowed by protraction of the long or diastolic pause. During this pause, blood continues to flow into the ventricles, slowly and passively, from the auricles, till, at its close, active auricular contraction takes place.

Aortic recoil is the reflex of left ventricular systole, is delivered abruptly and vigorously within the period of the second sound, and, being a purely physical phenomenon, it may be regarded as a fixed quantity, so long as the essential conditions of its development, vascular tension and elasticity, remain unaltered (see p. 91). Where incompetency of the aortic valves exists, the change of vascular tension thence arising, tends rather to abbreviate than prolong the period of aortic reaction, owing to the sudden emptying of the first portion of the vessel by reflux upon the ventricle. If, for the reasons already mentioned, quinine and strychnia be preferred, I give two grains of the former with three minims of the latter and three minims of dilute sulphuric acid, in an ounce of water sweetened with syrup of orange flowers, three times a day. Symptoms indicative of pulmonary congestion, viz., dyspnoea and feeling of thoracic oppression, cough, and hæmoptysis, should be treated by means of dry cupping, warm poulticing, and counter-irritation of the chest. Temporary engorgement of the liver and portal system, indicated by epigastric fulness and tenderness, with increased extent of hepatic dulness, constipation, flatulence, foul tongue, loathing of food and retching, and frequently also by enlargement and protrusion of the hæmorrhoidal veins, may be promptly relieved by the application of two to four leeches at the pit of the stomach,

followed by a warm fomentation or poultice; a few grains of blue pill or grey powder should also be given, and some hours subsequently, a saline aperient. During this temporary derangement of the digestive organs, thirst should be relieved by the smallest possible quantity of fluid, and the food taken should be exclusively liquid.

Dyspepsia in such cases is due, not to uræmia, as Dr. Milner Fothergill surmises,* but to impairment or arrest of absorption in the stomach, from congestion of the portal system. I have repeatedly witnessed the symptoms previously described, where the urine was passed in full quantity, and was normal in all respects save as to deposit of lithates, and where, moreover, none of the ordinary symptoms of uræmic poisoning existed. Even assuming, as must, I believe, be now conceded, that the formation of urea is effected in the liver,† it is scarcely conceivable that the urine should not, in the cases referred to, have afforded evidence of its non-elimination, by reduction of specific gravity; and that none of the nervous symptoms usually associated with retention of urea should have been exhibited. I regard intercurrent symptoms of angina pectoris, viz., substernal pain and constriction, paroxysmal dyspnoea, and faintness, as evidence of subacute inflammation of the aorta; and I treat them by the application of leeches to the sternum, and the exhibition of small and frequent doses of mercury with chalk, and antimonial powder, combined. Two grains of each may be given every second hour. These measures rarely fail to afford prompt and complete relief.

Edema is best treated by cupping or leeching, and subsequently poulticing the loins, with a view to relief of the renal congestion. The hot air bath may then be used with great advantage, followed by an active drastic purgative, such as half a drachm of the compound powder of jalap, or two drachms of the acid tartrate of potash in a wineglassful of infusion of senna. Digitalis, as previously directed, may be given with a view to the production of active diuresis; an object which it seldom fails to accomplish.

* *Lancet*, May 30th, 1874.

† *Vide Functional Derangements of the Liver*, by Dr. Murchison, 8vo., 1874, *passim*.

The following cases are submitted, as examples of the various forms of lesion of the aortic valves discussed in the preceding pages. Amongst them will be also found examples of valvular inadequacy from dilatation of the root of the aorta.

CASE LXXVII.—*Hypertrophy of the Left Ventricle ; Mitral Regurgitation ; Aortic Obstruction, and Incipient Aortic Regurgitation.*

Marianne O'B., aged eighteen years, a dressmaker, admitted into hospital, June 28th, 1865. The following history was obtained from her mother. When only six years old she was taken to bathe by a female friend, and was kept in the sea fully an hour. On that evening, she was attacked with acute rheumatism of the knees and ankles ; the illness lasted five weeks, and it would seem, from the imperfect account given, that the heart was implicated. A year subsequently she had another attack of rheumatism, and sought relief in the extern department of one of our city hospitals. She waited in an outer room, where, by some oversight on the part of the attendants, she remained unobserved, and was locked up. When discovered in the evening, after several hours of confinement, she was quite exhausted from crying, and was put to bed and treated with leeching and blistering over the heart. She was also, on this occasion, treated for threatened disease of the spine. She was twelve months under treatment, and was greatly reduced in strength.

The following was her condition when she came under my notice. The pulse was not calculable at the wrist, owing to its weakness and irregularity. The pulsations of the heart were 156 in the minute, and remarkably irregular. There was orthopnoea ; the precordial dulness was much extended transversely, and the cardiac impulse, strong and heaving, was accompanied by a jarring fremitus. At the apex, a loud rasping murmur accompanied and succeeded the first sound ; no second sound was here audible. Over the lower part of the sternum, and at the base generally, both sounds were heard ; the first, accompanied by a murmur which was softer than that heard at the apex, and the second, of a partially blowing character

The basic systolic murmur was transmitted through the arch of the aorta, and into the arteries of the neck. There was dulness of the bases of both lungs; and in these situations, and also under both clavicles, crepitant râles were audible. One leech was applied over the heart, and tincture of digitalis was given in \mathbb{M}_x doses, with camphor water, every third hour.

On the following day there was evidence of slight improvement. Pulse 126, irregular; heart's action less tumultuous; face slightly bloated, especially at the eye-lids; lips and cheeks somewhat livid; teasing cough when the patient attempted to lie down.

On the 30th there was still further improvement. Pulse 108, and stronger.

The digitalis was stopped on the 2nd July; and chloric ether, with tincture of orange peel, was given in the infusion of *calumba*.

July 5th. Pulse 96, and distinct, but irregular. Appetite now moderately good, and patient able to sleep comfortably in the recumbent posture. Physical signs as previously noted, except that the second sound was now audible at the apex, was double at the base, and was accentuated to the left of the sternum. A systolic murmur was audible all over the front and back of chest, and was propagated into the abdominal aorta as far as the umbilicus. No visible arterial pulsation, and now scarcely any cough. The medicine last prescribed was directed to be continued, and the patient was allowed to get up. These favourable changes were fairly attributable to leeching over the heart, and digitalis given as previously mentioned.

Shortly after this date, she left hospital, but was re-admitted on the 14th; having had, on the preceding night, a syncopal attack, in which it was thought she would have died. State on re-admittance: pulse not calculable at wrist; estimated by the heart, it counted 132. Physical phenomena as last reported, except that a musical note was now audible with the first sound over the lower sternum and likewise in the course of the aorta as high as the right second costal cartilage. It was heard with three or four consecutive systoles, and then, during as many more, it ceased to be audible. Digitalis, as previously given, was resumed.

She died on the 30th August ; examination of the body was not permitted. The diagnosis, however, was sufficiently clear. The musical quality of the aortic systolic murmur at the date of re-admittance was most probably due to a vibrating shred of fibrin appended to one of the segments of the aortic valve.

CASE LXXVIII.—*Visible Pulsation of Carotid and Temporal Arteries ; Double Murmur at Apex and at Base of Heart, and likewise in Left Axilla ; a Single Systolic Murmur in Carotids and in Descending Aorta. Death two years and nine months later. Autopsy: Pulmonary Apoplexy ; Left Pleural Effusion, General Adhesion of Pericardium, and Enlargement of Heart ; Hypertrophy, with Dilatation of Left Ventricle ; Thickening and Inadequacy of Mitral Valve ; Obstruction and Inadequacy of Aortic Valve ; Diminution in Calibre of Aorta.*

John L., aged fifteen years, a messenger, admitted March 6th, 1867 ; had never suffered from rheumatism, but at the age of eight years had scarlatina. Subject to cough for many years, and spat blood several times ; the date of the last hæmoptysis being Friday, 24th ultimo. Pulse 102, sharp, and abrupt, but not visible at wrists or in the temples. Bowels regular. No œdema. Precordial dulness extended to left. Apex-beat immediately beneath nipple, and extending over an area of one and a-half square inch in diameter.

8th. Pulse 102. A double murmur audible at apex, the systolic being the louder of the two, and of a blowing character ; it is likewise audible at base, and in course of ascending aorta ; but here the diastolic murmur is the louder, and *follows* the second sound, the latter being sharp, clear, and ringing over midsternum, and not accentuated over pulmonary artery. Neither murmur audible over left scapula, but both faintly so, and manifestly by transmission, in left axilla. A single systolic murmur is heard in cervical arteries. Respiration and respiratory phenomena normal ; has slight cough.

April 24th. Discharged much improved in general health.

The particulars of the case at that date were noted as follows ; viz., pulse of good volume, and ranging from 90 to 100. No respiratory distress. No congestion, œdema, or hepatic engorgement. Two distinct murmurs audible at midsternum, where they are likewise loudest. Of these, one is systolic in rhythm, and rough in quality ; the other is postdiastolic, soft, and blowing. Both are propagated through the aortic arch, and the former, likewise, into the great cervical arteries and thoracic aorta. Second sound not intensified in pulmonary artery. Strong arterial pulsation in neck ; and on right side pulsation also in internal jugular vein. Slight visible pulsation in temporal arteries, but not in radials. Treatment consisted in the use of the American wild cherry, given in form of infusion. The following note was made at this date, previously to the boy's discharge ; the diagnosis being aortic valve disease, not in an advanced stage, and unassociated with the usual consequences of the affection. " Valves partially competent, reflux taking place only after the act of closure, and giving rise to a murmur, postdiastolic in time, *i. e.*, immediately succeeding the second sound, which was sharp and clear ; thus indicating a condition of valves in which their *tensive* properties were not impaired, but their edges were probably tuberculated, and incompetent by want of coaptation."

On the 9th November, 1869, this patient, now a grown young man, was re-admitted for rheumatic pains in several joints. There was a visible pulsation in the carotid, temporal, and brachial arteries. Pulse at wrist sharp and collapsing, and arhythmically irregular. Apex-beat in nipple line ; and here, a fremitus was perceptible. Here, likewise, a double impulse was felt, and a substitutive systolic bellows-murmur was audible. At base a double murmur was heard ; viz., a systolic, which was likewise transmitted into the carotids, and a soft and blowing murmur *succeeding* a sharp second sound.

December 29th. After a fortnight's absence from hospital duty, I found him much emaciated, and expectorating blood-stained mucus. Respiration embarrassed. Precordial dulness extended. He complained of an overpowering sense of oppression across the chest. A blister was applied to the front of the

chest, and some antispasmodic medicine was given. On the following day he felt much relieved. Pulsation was then slightly visible in the radial arteries, and the second murmur at the base was rather diastolic and substitutive, than postdiastolic in rhythm. No œdema.

January 4th, 1870. Over border of left costal cartilages, in nipple line, and to a distance of two inches around this point, as likewise over entire epigastrium, a loud metallic tinkle was heard synchronously with the double impulse of the heart. It was loudest in the first mentioned situation, and was audible during expiration only; the descent of the diaphragm and inflation of the lung seeming to suspend or mask it, probably by separating the pericardium and stomach.

12th. He died suddenly, and apparently of syncope, during an effort of vomiting.

Examination of the body twelve hours after death. A few nodules of extravasated blood were found in the lungs. The left lung was compressed by serous effusion into the pleura, and its surface was opaque and corrugated. The pericardium was thickened and universally adherent to the heart; the false membrane by which it was attached, was two lines thick, and presented a blood stain posteriorly. The heart was greatly magnified and elongated, and, with half an inch of the great vessels attached, it weighed twenty-eight and a-half ounces. Right chambers normal. In the right ventricle there was a thick flake of yellow fibrin connected with the tricuspid valve, and extending, in the form of a flattened cylinder, into the pulmonary artery. Left auricle thin; appendix bound down to surface of auricle by adhesion. Anterior right segment of mitral valve presented three wart-like excrescences on the auricular aspect of the free margin, and in the vicinity of these, the valve was slightly thickened. The remainder of this valve, as likewise the entire of the posterior segment, was sound. The walls of the left ventricle were an inch thick at the base, and its cavity was much dilated and elongated. A thin flake of fibrin adhered to the septum. The aorta was reduced in calibre to the size of the middle finger. Its lining membrane was of a scarlet tint, and its valve-segments were shrivelled or rolled up at the free edge; but structurally

they were apparently healthy. When approximated by means of water poured into the aorta, they left a large opening in the axis of the passage, through which the water rapidly flowed into the ventricle. The aorta and its primary branches contained some dark blood-clot.

In reference to this case, I wish only to observe upon ; (a) the upward displacement of the point of apex-pulsation, in connexion with general adhesion of the pericardium ; (b) the existence of a basic *postdiastolic* murmur, where incompetence, without structural disease of the aortic valve existed, and the subsequent conversion of this into a veritable diastolic murmur, at a time when, presumably, the valve was beginning to undergo structural change ; (c) the absence of accentuation of the second sound in the pulmonary artery, and of audible murmur at the left scapula, where veritable mitral reflux existed ; (d) the existence of only a systolic murmur in the carotids, where a double murmur was audible at the base, and in the arch of the aorta ; and, (e) the existence, at the precordium, of a metallic tinkle communicated from an inflated stomach, and assuming the rhythm of the double cardiac impulse.

CASE LXXIX.—*Anasarca and Albuminuria ; Extension of Precordial Dulness Transversely ; Feeble Impulse of Heart, and Basic Systolic Murmur transmitted into Aorta and Carotids ; Death. Autopsy : Effusion into the Pleuræ and Pericardium ; Hypertrophy and Dilatation of Left Ventricle ; Obstruction and Incompetency of Aortic Valve ; Renal Disease.*

James A., aged twenty-six years, a car-driver, admitted June 4th, 1867. Two months previously he had a chill, which was followed by cough and expectoration. At the date of admittance he was remarkably pallid, the lower limbs and trunk were swollen, and there was cough with expectoration of rust-coloured mucus. Respiration 48. Pulse 120, and dicrotic. Throbbing of carotids. Slight congestion of lips. The area of precordial dulness was increased transversely. No perceptible cardiac impulse. At the apex of the heart, a feeble and distant systolic murmur was audible, and the second sound was faint, but other-

wise normal. Over midsternum, a loud systolic bellows-murmur was heard; it was likewise audible in the arch of the aorta, and faintly in the carotids. Crepitant râles were audible all over the right side. Urine passed in small quantity, 1·015 in sp. gr., loaded with albumen, and containing numerous red-blood corpuscles, and a few hyaline tube-casts to which some epithelium still adhered.

9th. Patient pale and somnolent. Precordial dulness increased in extent. Heart-sounds obscure, and no murmur to be heard. Comparative dulness at postero-inferior portion of right side, where crepitant râles are audible.

10th. Died early this morning. *Post mortem* examination of body *vesperi*. Copious effusion of serum into both pleuræ and pericardium. Heart greatly enlarged transversely, and by hypertrophy of the left ventricle exclusively; right chambers normal, and containing some dark coagulated blood; left ventricle much dilated and thickened. The mitral orifice and valves were normal and competent. All three segments of the aortic valve presented, at their free edge, a crop of rough granular vegetations, which extended to a considerable distance on the ventricular surface of the valves, so as necessarily to obstruct the exit of blood from the ventricle. The left anterior segment presented a rent or opening, as large as a pea, near its free margin. The edges of this rent hung into the ventricle, and were coated with granular lymph, forming a tubular passage about half an inch in length, which permitted regurgitation, and at the same time, by its prominent margins, obstructed the orifice of the artery. The edges of the valves, moreover, hung loosely into the ventricle, and permitted free regurgitation. The substance of the heart was firm. The aorta was healthy. Liver and spleen greatly enlarged and congested. Kidneys enlarged, their cortex thickened, and presenting a light fatty appearance. By an oversight they were not examined microscopically. The lungs were examined, but no note was made of their condition.

CASE LXXX.—*Rheumatic Fever; Œdema; Weak and Intermittent Pulse; Paroxysms of Violent Palpitation and Cardiac Asthma, with Extreme Rapidity of Pulse; Jugular Pulsation; Enlargement of Liver, and Ascites; Suppression of Second Sound of Heart; Systolic Murmur at Apex, and Double Murmur at the Base of the Heart.*

Owen G., aged twenty-three years, a farmer, admitted into hospital, September 4th, 1867. Has worked hard as an agricultural labourer. Eight years since he had measles, and two years subsequently, rheumatic fever after a severe wetting. Shortly after the last mentioned illness he began to suffer from palpitation and shortness of breath on exertion. He likewise had occasionally œdema of the feet, which passed off with critical diarrhœa and diuresis.

When admitted, he was suffering from general cedema, dyspnoea, and palpitation. The urine was moderate in quantity, 1·025 in sp. gr., acid, and free from albumen and sugar. Copious diaphoresis. The hair of the head was erect, and of a spinous character. Precordial dulness extended horizontally from the left nipple line to the right of the mesial line of the sternum, its vertical extent being normal. A loud systolic bellows-murmur was heard below the nipple, and likewise, but less distinctly, over the base, and at the xiphoid cartilage. A second sound was nowhere audible. The supraclavicular fossæ were obliterated, and on the right side there was visible pulsation of the external jugular vein, which was synchronous with the impulse of the heart, and more distinct with inspiration than with expiration. The chest was prominent on both sides, and on the right there was considerable dulness below the clavicle, and all over this side loud râles were audible. Over the left side, on which he seldom lay, respiration was puerile. The liver was enlarged, and tender to pressure; and there was some liquid in the peritoneum. The radial pulse was slightly visible, weak, and intermittent. The intermission recurred at unequal intervals; thus, it was noted after six, eight, nine, and fourteen pulsations, respectively. On the 9th September it occurred four times in succession, after

seven, eight, and nine beats respectively; then after thirty-two beats; and next, four times successively after fourteen beats.

R. Infusi pruni Virginianæ, ʒviiss; Spirit. æther. nitros., ʒiv. A tablespoonful to be taken every fourth hour. To have saline aperients as required; nutritious diet; and a moderate allowance of wine.

On making my morning visit on the 10th September, I found him suffering from an aggravated paroxysm of cardiac asthma; the heart was acting tumultuously, and at the rate of 198 pulsations in the minute; respiration 48, and the body bathed in perspiration. No cardiac murmur was then audible. This paroxysm occurred without any assignable provocation. To have immediately a draught composed of Hoffman's anodyne, ʒss; Battley's sedative, ʒj; and camphor water, ʒj.

On the following morning (11th) the pulse was only 98, regular, and without intermission.

16th. It was noted that since last report he had had one paroxysm, but of a less severe character than the first. The pulse had varied from 96 to 108, and been occasionally free from intermission. The sedative draught last mentioned had been continued, and a pill had been given daily, consisting of equal parts of blue pill, dried soda, and compound rhubarb pill; and on that morning, owing to the existence of unusual hepatic tenderness, a single leech was applied over the liver, and subsequently a warm poultice.

17th. 11 A.M. I found the patient again in a paroxysm. Pulse 198, and respiration 54. No murmur detectable. Sedative draught repeated.

18th. Complained of "swimming" in the head when he dozed. Pupils small; pulse 96, regular, and not intermittent. Tincture of hops (ʒj) was substituted for opium in the draught.

23rd. 10 A.M. Patient sitting up in bed, face flushed, and body bathed in perspiration. Pulse failing, and not calculable at the wrist; but registered by the heart, it was 210 in the minute. The right external jugular vein and its tributaries were greatly distended. To have former sedative, but without opium.

He passed a good night on the 24th, and on the following morning the pulse was 108 and regular. Considerable cedema of

left hand and forearm, notwithstanding that decubitus is usually *dexter*. No second sound audible.

27th. A double murmur was audible at midsternum, to the left of the median line; and a faint but rough second sound was heard in the course of the aorta. Pulse 96, steady, and regular. Much œdema of left hand and forearm.

October 5th. Patient flushed, and perspiring. Pulse 192; respiration 36. This paroxysm, which came on during sleep, was of brief duration. He passed a good night, and on the following morning (6th) the pulse was only 84.

November 8th. 11 A.M. Pulse 210, counted by the heart; respiration 48, and accompanied with great dyspnoea and inability to speak; face flushed and perspiring. The entire chest was visibly shaken by the violent action of the heart.

On the evening of this day, the patient abruptly left hospital, and I have not since heard of him.

The details of this case, even in the absence of *post mortem* evidence, are given at great length, because of the typical character of the cardiac asthma, and the extreme rapidity of pulse which it exhibits. I have met with only two cases, of which this was the first, in which the rate of cardiac pulsation reached 200; in this instance, on two different occasions, it exceeded that number by 10 beats. The diagnosis implied in the title of the case is, I venture to think, warranted by the symptoms and signs exhibited.

CASE LXXXI.—*Double Basic Murmur, remarkably Loud and extensively Transmitted; History of Sudden Occurrence of Cardiac Lesion from Strain. Diagnosis: Yielding of Segment of Aortic Valve previously unsound.*

Richard W., aged thirty-six years, a labourer of intemperate habits, was admitted, December 29th, 1868. Had been for some time subject to cough; and six days prior to admittance, whilst carrying two buckets full of liquid food to cattle, he felt a sudden yielding in the region of the heart, which was followed by a buzzing noise distinctly audible to himself. He felt no pain in the chest, and had no paroxysm of dyspnoea either then or

since. There has not been hæmoptysis. The buzzing noise in the chest has continued up to the present, and in bed it has been particularly loud and troublesome.

The appearance of the man indicates good health. There is, however, shortness of breath with orthopnoea, but no œdema. Pulse in erect posture 108, regular, and equal on the opposite sides, but visible; in the recumbent posture it is 84. Chest universally resonant, and respiration natural.

In the erect posture a fremitus is perceived on placing the hand over the precordium; the seat of this fremitus is basic. Precordial dulness normal in extent. Apex beat in fifth intercostal space and in nipple line, and perceptible over an area of one inch in diameter. Here both sounds are accompanied by murmur, manifestly transmitted; the first, or systolic, soft and blowing, and the diastolic, jarring and faintly musical.

At midsternum these murmurs attain their maximum intensity, and replace the normal sounds of the heart; the systolic, loud and blowing, and the diastolic, loud, buzzing, and musical. Both murmurs, retaining the characters just described, are diffused over the entire front of the chest, but with a loudness progressively diminishing in proportion to the distance from midsternum. The diastolic murmur is likewise audible in the carotid and brachial arteries, in the abdominal aorta and iliac arteries, and faintly also over the lumbar spine. In the recumbent posture, and after repose, it loses its musical character, and the fremitus is then not perceptible at the precordium.

After a short residence in hospital, this man returned to his employment, somewhat improved by rest. I have not since heard of him. The features of special interest in the case are: the absence of sub-sternal pain, dyspnoea, and hæmoptysis at the time of the accident, assuming it to have been of the character diagnosed; and the transmission through the principal arteries of the second order, and to a great distance, of an aortic *diastolic* murmur. In both these respects the case is, in my experience, unique.

CASE LXXXII.—*Repeated Attacks of Acute Rheumatism; Double Cardiac Impulse, and Double Murmur of Aortic Origin; a Systolic Murmur audible in the Arteries of the Neck, and a Diastolic Murmur in the Thoracic and Abdominal Aorta.*

Thomas B., aged twenty-two years, an accountant, visited hospital as an extern, January 30th, 1874. Had acute rheumatism seven years previously, and a similar attack each succeeding winter since that date. Has smoked and drunk to excess, but for the last six months has been temperate in both these respects. Has not had hæmoptysis. Is pale and nervous; breathing natural. Pulse 108, and remarkably variable as to rate independently of physical effort, and visible in carotid, temporal, and radial arteries. Can lie on left side. There is strong, diffused, and double cardiac impulse; no localized apex beat. At midsternum there is a double murmur, the systolic element of which is of a blowing character, and ends with a sharp and clear valve-click, the diastolic being much louder, and also blowing. Both murmurs are audible all over front of chest, and distinctly below and outside left nipple line. The systolic murmur alone is heard in the carotid arteries, and the diastolic only, but very distinctly, along the dorsal and lumbar spine. To have tincture of the perchloride of iron, and spirit of nitrous ether, of each ℥x, with ℥v of tincture of digitalis, in an ounce of infusion of quassia, thrice daily.

The diagnosis is given in the title of the case, and no comment upon it is necessary; except that it exemplifies still further the truth, so often previously insisted upon, that whilst aortic murmurs of diastolic rhythm are extensively transmitted through the main trunk of that vessel, they are *not* audible in its primary branches; whilst aortic systolic murmurs of valvular origin *always* are audible in these vessels. Case 81 affords a notable exception to the former statement. The following case still further illustrates the preceding remarks.

CASE LXXXIII.—*Double Aortic, and Systolic Mitral Murmur. Diagnosis: Aortic Obstruction and Reflux, and Mitral Regurgitation.*

James O'B., labourer, aged thirty years, seen as an extern,

May 28th, 1869. Has been three years in the army, but never in active service, and retired three years ago. Has had palpitation for the last ten years; but his health failed only at Christmas last. Pulse 84. Visible pulsation of carotid, temporal, brachial, and radial arteries. Respiration embarrassed. Decubitus *dexter*. Cough, with copious expectoration. Engorgement of both lungs posteriorly. No œdema anywhere. Cardiac impulse heaving, and apex-pulsation diffused.

A double murmur was audible at the base and in the ascending portion of the arch of the aorta, the diastolic being the louder, and not audible in the neck; whilst the systolic murmur, which became fainter in the ascending portion of the arch, was exaggerated in the carotids, and was there attended with fremitus.

Immediately to the right of a vertical line two inches inside the left nipple, the systolic murmur was faint; but to the left of that line it was louder, and thence to the site of apex-pulsation in the nipple line and sixth intercostal space, it gradually increased in intensity. The second sound in the pulmonary artery was not accentuated, and no murmur was audible in the back. To have tincture of the perchloride of iron, chloric ether, and ipecacuanha wine in camphor water.

The following case is remarkable in regard to its duration, its apparent gravity being considered.

CASE LXXXIV.—*Dyspnœa ; General Venous Congestion and Œdema ; Extreme Weakness and Irregularity of Pulse ; Mitral and Aortic Regurgitation.*

Bridget M., a domestic servant, aged twenty-six years, admitted into hospital in the early part of 1868, and seen again as an extern on the 26th October in that year. Respiration much embarrassed after exertion, and oppressed even when she is at rest. Pulse remarkably weak and irregular, and not calculable at the wrist. Action of heart irregular and intermittent. Œdema of feet, and congestion of lips. At the apex of the heart there was a systolic murmur, and at the right base a soft dia-

stolic murmur replaced the second sound, which was audible only in the pulmonary artery.

This poor girl was re-admitted in July, 1872, in the same condition; she left, relieved, after a month's rest and tonic treatment.

CASE LXXXV.—*Systolic Murmur at Apex; Double Murmur at Base; Systolic Basic Murmur extensively transmitted through Arteries. Diagnosis: Mitral Regurgitation; Aortic Obstruction and Regurgitation.*

Henry H., aged thirty-six years, a labourer, admitted July 1st, 1867. Health reported good up to six months ago, when, without having been ill, or having sustained injury of any kind, he began to suffer from pain in the left side, and lower part of the back. Two months subsequently he observed a swelling in the left side of the abdomen, and in the site of this swelling he had occasionally pain of a spasmodic character. Urine high coloured, and depositing amorphous lithates. Pulse 68, and "collapsing." Visible pulsation in carotid, radial, and femoral arteries. Strong pulsation of abdominal aorta, accompanied by a purring tremor and a systolic jarring bruit. A systolic bruit of a somewhat rough character existed at the apex of the heart, which pulsated in the sixth intercostal space, one and a-half inch outside the nipple line; it was audible over an area measuring one and a-half inch transversely, by one inch vertically. This murmur was likewise heard in the left axilla and left back. At the apex, the second sound was not distinct.

At midsternum, a systolic and a diastolic murmur existed; both were transmitted in the course of the ascending aorta, and were loud, soft, and blowing, but the diastolic was the louder of the two, and superseded the second sound. The systolic murmur was propagated into the carotids, abdominal aorta, and femoral arteries. Second sound not exaggerated in the pulmonary artery. No dyspnoea. Discharged somewhat relieved, July 14th.

CASE LXXXVI. — *Systolic and Diastolic Basic Murmur, the former accompanying, the latter substitutive; Reduplication of Second Sound. Diagnosis: Obstruction and Regurgitation at Aortic Orifice, and Considerable Dilatation of Left Ventricle.*

James H., a stucco-plasterer, temperate, aged forty-four years, admitted into hospital, September 27th, 1869. Has never had rheumatism, but has had flying pains in various parts of the body. During the last six months has been weak, and often, within that time, has nearly fallen down in the streets from faintness. Sight began to fail, especially in the right eye, three weeks ago, and is now very much impaired; the right pupil is clear, but scarcely responds to light. Patient remarkably pale. Respiration tranquil; decubitus indifferent. Pulse 78, bounding and visible. No cedema. Apex of heart pulsates half an inch outside nipple line, in the fifth intercostal space, and over an area of one inch in diameter. Here both cardiac sounds are obscure, a faint transmitted murmur accompanying the second sound. At midsternum, and also in the course of the ascending aorta, a double murmur is heard, the systolic being the less distinct, and accompanying, whilst the diastolic murmur is loud, blowing, and substitutive. The former is transmitted into the carotid arteries, but the latter is not. No murmur audible in back. Second sound normal as to quality, and not accentuated in the pulmonary artery; it is double throughout the precordium, the first element being sharp and clicking, the second element represented, as already stated, by a loud bellows-murmur, which is most distinct at base, and masked and prolonged at apex. R. Tinct. ferri perchlorid., æther. chloric., aa. ℥x tertia, q. q. horâ s.

On the 9th October, which is the date of latest report, the second sound was not anywhere audible. The man left hospital soon afterwards.

The coexistence in this case, as in others to be afterwards noticed, of aortic regurgitation with double second sound, enabled me to identify with certainty the aortic and the pulmonic elements of that sound, respectively. The former, which was

posterior in time, was represented by a murmur; whilst the latter was sharp and clear. I believe the relative position in time of the two factors of the second sound, in this and all similar cases, may be accounted for by the existence of dilatation of the left ventricle, owing to which the evacuation of that chamber, and therefore the reaction of the aorta, are necessarily postponed.

If this view be correct, it follows that the existence of double second sound, in connexion with the murmur of aortic reflux, indicates dilatation of the left ventricle.

CASE LXXXVII.—*Rheumatism, Cough, and Hæmoptysis; Orthopnœa and Paroxysmal Dyspnœa; Congestion of Liver, and General Edema; Visible Pulsation of Arteries; Increased area of Precordial Dulness; Feeble Impulse; Double Murmur at Base, and a Single Musical Systolic Murmur in Aorta and Arteries of Neck, and likewise audible along the Spine; Gangrene of Feet; Hæmoptysis; Death. Autopsy: Heart greatly Enlarged, and Laden with Fat; Aorta Dilated and Atheromatous, and its Valves Incompetent by Dilatation of the Vessel, but also slightly Diseased and presenting Obstruction to Exit of Blood; other Valves healthy; Right Ventricle Thickened, but not Dilated; Left Ventricle Dilated and Thickened.*

John C., aged forty years, admitted November 16th, 1869. Has served twenty-one years as a soldier, ten of which were passed in India. Had an attack of rheumatism eight years ago whilst in camp at Shorncliffe, and has had cough for the last twelve months. Left the army last May. A fortnight ago he spat blood for two days, and about the same time his feet began to swell. For the last four months he has not been able to lie down, owing to the immediate occurrence of urgent dyspnœa on his attempting to do so; and for the latter half of that period he has been subject to paroxysms of dyspnœa, coming on suddenly whilst he lies quietly propped up, and obliging him to start out of bed to avoid being suffocated.

State on admittance: feet, legs, and thighs greatly swollen,

and very tense, but pale and waxy-looking. Scrotum also cedematous. Toes congested; face of an olive tint, but not jaundiced. Liver enlarged and tender. Pulse 96, and visible in the carotid, temporal, and radial arteries. Precordial dulness much extended, but cardiac impulse diffused and feeble; no distinct apex-pulsation to be felt. At base, a systolic and a diastolic murmur existed; the former musical, and transmitted through the aorta to its bifurcation, and likewise into the carotid and subclavian arteries; it was audible along the dorsal and lumbar spine, and, by transmission, at the apex. The diastolic murmur was soft, and confined to the base and the ascending aorta. Congestive enlargement and tenderness of the liver. To have an aperient, and afterwards a mixture of tincture of the perchloride of iron and chloric ether, in camphor water. To have, at night, a sedative, composed of Hoffman's anodyne and liquor of hydrochlorate of morphia. Feet and legs punctured, and yielded a copious discharge of serum.

26th. Lower limbs now enormously swollen, red, and tender, and on dorsum of right foot, incipient bullæ, indicative of proximate gangrene. Interval between cardiac impulse and radial pulse quite distinct, the pulse-rate being 96. The stroke of the radial artery coincided with the second sound of the heart; whereas, in the absence of organic disease of the heart and aorta, as I have ascertained by experiment, this does not occur till the rate of cardiac pulsation has attained to 126 in the minute.* Patient insists on having the window opposite his bed kept open day and night; and to admit of this being done without danger to the other patients, his bed was removed to a window at the end of the ward, and there enclosed on three sides by means of screens. Is spitting blood.

28th. Died this morning. The feet had previously assumed a dark chocolate hue, and several gangrenous bullæ had formed upon them. For some time before death the patient had ceased to crave for fresh air, and desired that the window should be closed.

* *Vide* paper "On the Rhythm of the Heart," Proceedings of the Medical Society of the College of Physicians, *Dublin Quarterly Journal of Medical Science*, August, 1864. This is the only example of veritable postponement of the radial pulse which I have met with. See p. 832.

On the following day, the heart, which was the only organ I could procure for examination, was carefully inspected. Emptied of blood, and with an inch and a-half of the aorta and pulmonary artery attached, it weighed twenty-seven ounces; it was of a globular figure; the apex was blunted, and the base was extensively and deeply covered with fat. The aorta was dilated above the valves, and readily admitted three fingers introduced edge-wise. The valves were incompetent, water poured into the aorta readily flowing into the left ventricle. The line of union of two of the valves was hard and cartilaginous, and offered an obstacle to the efflux of blood from the ventricle. Otherwise, the valves were healthy. The lining membrane of the aorta presented several yellow patches of incipient atheromatous change. All the other valves were normal and competent. Right ventricle somewhat thickened, but not dilated. Left ventricle dilated, and three-fourths of an inch thick at base and centre. Muscular structure healthy.

CASE LXXXVIII.—*Scarlatina and Aortic Valvulitis; Extensively transmitted Aortic Systolic Murmur. Diagnosis: Aortic Obstruction, Embolism, and Recurrent Right Hemiplegia.*

Thomas B., aged seven years and a-half, a fine healthy looking boy, was admitted into the Mater Misericordiæ Hospital, under my care, on the 5th of February, 1874.

Had scarlatina three years previously. Three months prior to date of admittance, he complained of "pain in the stomach," which quickly passed away; nine weeks later he suddenly lost the use of the right arm, and, partially, that of the right leg; articulation was likewise affected.

From this attack, it would seem that he quickly recovered. On the 4th of February (the day preceding that of admittance), he was again suddenly deprived of the use of the right arm and leg.

When I first saw the boy, on the 6th, he was entirely powerless as to the upper and lower limbs on the right side. Sensation was unaffected, as was likewise articulation, but reflex

irritability was defective in the paralyzed foot. Pulse 84, and regular. Apex-pulsation in left nipple-line. At the right base a loud *bruit de râpe* was heard with maximum intensity, and from this point it was transmitted through the aorta, quite to the lumbar spine, and into the carotid and subclavian arteries; it was likewise audible, but less distinctly, at the apex. The second sound was remarkably sharp in the aorta, and double during *inspiration only*. To have a teaspoonful of syrup of the phosphate of iron three times daily.

The boy is still (February 24th) under treatment. He has gradually recovered the use of the arm, which he can now lift to his head; but in the use of the leg very little improvement has been effected.

Tested by means of the æsthesiometer, sensibility is undiminished in the paralyzed limbs, whilst the dynamometer registers a pressure equal to twelve and a-half pounds for the paralyzed hand, and a pressure of twenty-seven and a-half pounds for the unparalyzed hand, as reported by my colleague Dr. Nixon. To him I am also indebted for the subjoined tracing of the pulse (Fig. LII.), taken on the 24th February. It is characteristic of obstruction, without regurgitation, at the orifice of the aorta, and shows also some degree of hypertrophy of the left ventricle.

It exhibits shortness and want of verticality in the up-stroke, and roundness of summit.

FIG. LII.



Thomas B.

Aortic obstruction. Pulse 78.

Before I had the advantage of seeing this tracing, I concluded from the position of the apex of the heart, and the force of the impulse, that the left ventricle was hypertrophied. From this I further inferred that the lesion at the aortic ori-

fice was of some years' standing, and had most probably originated in acute endocarditis, engaging the valves, at the date of the attack of scarlatina, three years previously ; and, finally, that embolism, by detachment of a flake of fibrin from the aortic valve, and its impaction in one of the arteries of the left cerebral hemisphere, most probably the middle meningeal, was the cause of paralysis on both occasions.

On the first occasion, the embolus was most probably composed of detritus, was quickly disintegrated by the force of the arterial current, and washed away from the obstructed vessel ; hence, the rapid recovery which followed. In the second attack, however, there is reason to believe that the plug was of a more compact and solid character, but is now undergoing a slow process of reduction by molecular detachment from the surface.

That the structural lesion causing a murmur of exit is confined to the root of the valve, does not involve its effective portion, and is chiefly of the nature of a deposit from the blood, I judge from the clearness of the second sound, the absence of diastolic murmur, and the harsh and grating quality of the systolic murmur, taken in conjunction with the actual occurrence of embolism.

Hemiplegia from this cause in childhood is of favourable augury, but very liable to recurrence.

The treatment required is of the simplest ; but excitement and physical effort, and, indeed, all circumstances whatever which tend to hurry the circulation, should be deprecated.

The absence of muscular contraction or cramps in the paralyzed limbs, and of impairment of sensibility, are worthy of notice, as characteristic of this form of paralysis.

Reduplication of the second sound during *inspiration only*, is not new to me. From the reduplication, I infer that the left ventricle is also dilated. The limitation of this phenomenon to the period of inspiration may, I think, be attributed to a virtual exaggeration of the inequality of capacity existing between the two ventricles at that moment, owing to the facility for disgorgement presented to the right ventricle during the inspiratory effort, whilst no corresponding influence is exercised upon the left ventricle by that act.

CASE LXXXIX.—*Recurrent Rheumatism ; Pericarditis ; Aortic Obstruction and Reflux ; Basic Systolic, and Postdiastolic Murmur ; Transient Systolic Murmur at Apex.*

William G., aged eighteen years, assistant in a wine store, was admitted into hospital, October 25th, 1869, suffering from rheumatic inflammation of the knees, feet, and right wrist, with which he was attacked on the 20th. Was under my care in hospital for a previous attack of rheumatism, complicated with pericarditis, from the early part of the preceding February, to the 17th of March following.

State on second admission. Is remarkably pale. Pulse 72, and scarcely visible. Apex-beat in the nipple line ; a loud, "whiffing," systolic murmur is audible at the apex, and is likewise transmitted to the left axilla and back. At the base, a faint systolic, and a loud postdiastolic murmur are heard. Of these, the former alone is transmitted into the carotid arteries. To have watery extract of opium, gr. j, thrice daily.

November 11th. To continue pill at night only, and take sulphate of quinia, gr. iss, twice daily.

25th. Discharged cured as regards the articular inflammation, but with the cardiac signs unaltered.

Admitted for the third time, April 21st, 1873, with a mild attack of rheumatism of the feet and legs, unaccompanied by swelling. Since date of last report, he has been employed as an engraver on wood. Actual condition : Pulse 96, full, regular, and visible. Precordial dulness extended to the left. Apex beat strong, and perceptible over an area of one inch immediately external to the nipple line. At the apex-point a soft systolic murmur is heard ; this is not audible in the left axilla or back, the second sound being faint, and associated with a feeble distant murmur. In proportion as the stethoscope is slowly shifted upwards and inwards towards midsternum, both these murmurs become gradually more distinct ; at the last mentioned point they attain a maximum loudness, which is maintained, with only slight diminution, through the ascending aorta. The diastolic murmur in both situations is the louder of the two, is soft and blowing, and preceded by a sharp second sound ; whilst

that of systolic rhythm is rough, and alone audible in the arteries of the neck. To have a quinine mixture thrice daily, and gr. j of watery extract of opium at night.

The existence of an indubitable mitral systolic murmur, of temporary duration, at the date of the second admittance, I would venture to attribute either to the accident of mitral thrombosis, or to temporary asthenia of the walls of the left ventricle. I cannot admit *cure* of an actual lesion of the valve, of which I have never seen presumptive evidence, much less proof. During the interval of more than three years between the second and third admittance, disease of the mitral valve, previously established, would have made such progress intrinsically, and would have entailed collaterally such an amount of structural alteration and functional impairment, as would have rendered the diagnosis of it a problem of the simplest character; Whereas, not only had no such changes been effected in the interim, but murmur had ceased to be audible at the apex, except by transmission.

The postdiastolic rhythm of the second murmur suggests to me marginal lesion of the valve only, and that of a trivial character.

The following case affords another example of a murmur of this rhythm, but due to a different and a more formidable lesion.

CASE XC.—*Rheumatism; Dilatation and Atheroma of the Aorta; Harsh Systolic Murmur, accompanied by Fremitus at the Base, and transmitted through the Arch of the Aorta, and into the Arteries of the Neck; also a Soft Postdiastolic Murmur at the Base, and transmitted through the Arch of the Aorta only.*

Michael B., aged fifty-five years, a man of a respectable class, but ruined in circumstances, and desponding, called on me, January 14th, 1870. Had lived rather freely, and nine years previously had rheumatism. Six months prior to the date of his visit to me, he began to suffer from a feeling of discomfort about the heart, with occasional palpitation, and also from vertigo and failure of memory. When I first saw him, he was anæmic; the face was puffed, and of an olive tint. Pulse 96, full, soft, arhyth-

mically irregular, and barely visible. Cardiac impulse very feeble, perceptible half an inch outside the nipple line, and beneath the sixth rib. At the apex, a transmitted systolic murmur was audible. At the base and in the course of the aorta, a very loud systolic bellows-murmur, and a faint postdiastolic murmur associated with a clear second sound, were heard; of these, the former only was transmitted into the carotids. No *arcus senilis*.

February 4th. A loud, harsh, substitutive systolic murmur, accompanied by fremitus also, a soft postdiastolic murmur existed at midsternum; both were transmitted throughout the arch of the aorta, but most distinctly so to the level of the second right costo-sternal articulation. Beyond the left sterno-clavicular articulation the diastolic murmur was not audible; neither was it to be heard in the arteries of the neck; whilst the systolic murmur was heard in this situation with remarkable distinctness. The latter was likewise audible at the apex, but here it was accompanied by a first sound. To have a mixture of iron and chloric ether in camphor water.

I conclude that the aortic valve in this case was extensively disorganized at the root; whilst, as I infer from the existence of a clear second sound, and from the harsh quality of the systolic murmur in the aorta, conjoined with the faltering and irregular character of the pulse, the inadequacy of the valves was due to dilatation of the aorta, involving the orifice.

CASE XCI.—*Palpitation; Sudden and Severe Epigastric Pain, followed by Vomiting and Collapse; Visible Pulsation in the Carotids; Double Impulse; Displacement of the Apex Outwards; Double Basic Murmur Transmitted into Aorta, Systolic Murmur alone being Audible in the Neck; Death. Autopsy: Passive Effusion into the Pericardium; Enlargement of, and Fatty Accumulation upon the Heart; Hypertrophy and Dilatation of the Left Ventricle; Interfascicular Fatty Deposition; Rupture of one of the Segments of the Aortic Valve, and Calcareous Transformation of its Root; Dilatation and Atheroma of the Aorta.*

Anne McE., aged forty-four years, admitted January 26th,

1870. Never had rheumatism. Six months previously, she experienced palpitation for the first time, and on the day prior to date of admittance, was seized with pain in the epigastrium, which was succeeded by vomiting. When admitted, she was still suffering from irritability of stomach, the ejecta consisting of the liquids drunk, mixed with mucus and bile. She was then in a state of collapse, chilled and pallid, the eyes sunken, and the breath cold; the whole aspect being that of a person in the collapse stage of cholera. The pulse was 102, very weak, but regular. Arterial pulsation was visible in the neck, but not in the temples or wrists. Apex-beat half an inch outside the nipple-line, but in the fifth intercostal space; and here a feeble second impulse was felt, and a transmitted systolic murmur was audible.

At midsternum, and in the course of the ascending aorta, a double murmur was heard, that of diastolic rhythm, which was blowing and substitutive, being the louder of the two, whilst the systolic murmur alone was transmitted into the arteries of the neck. To have spirit of chloroform, and hydrocyanic acid draughts.

Died in the course of the same day. About eight ounces of serum were found in the pericardium. This was the product of simple passive effusion, as no trace of pericarditis existed; the serous surface being smooth and transparent. The heart was enlarged, weighing twelve and a-half ounces, and remarkably soft and flabby, expanding by its own proper weight when laid upon the table. There was a good deal of fat on the surface. Examined under the microscope, the muscular fibres exhibited normal striation, and seemed healthy; their diameter varying from 1·776 to 1·332 part of an inch; but between the fasciculi there was a good deal of oil deposit.

The left ventricle was somewhat dilated and thickened. The aortic semilunar valves were healthy at the free borders, and competent to close the axis of the passage; but one of them was ruptured in the centre, the rent admitting the point of the little finger, and permitting free reflux into the ventricle when water was poured into the aorta, whilst the edges of the valves came into close mutual apposition. The root of the ruptured valve

was calcareous, and two rugged masses of chalk-like substance, as large as peas, projected on the ventricular surface of the valve, and must have offered considerable obstruction to the outgoing current. The aorta was dilated above the valves, admitting the points of three fingers, and exhibited atheromatous mottling in an early stage.

The legitimate inference from the history of the patient's last illness and from the actual state of the heart as found after death, is that rupture of the valve-segment took place on the day preceding her demise, and that the shock thereby induced, operating upon an already feeble heart, was the immediate cause of collapse and death.

CASE XCII.—Rheumatism ; Fright followed by Palpitation, which became Habitual ; Pulmonary Emphysema and Edema, with Cough, Dyspnœa, and Congestion of Face and Neck ; Visible Pulsation of Superficial Arteries ; Displacement of Apex of Heart Outwards and Downwards ; Substitutive Systolic Murmur at Apex, audible likewise in Left Axilla and Back, with Faint Second Sound ; Double Murmur at Base. Diagnosis : Aortic Obstruction and Reflux, and Mitral Regurgitation.

James C., aged twenty-eight, a cabman of large stature, and intemperate, admitted February 5th, 1870. Six years previously, had rheumatic pains in legs and arms, for which he was treated in the Hardwicke Hospital for six weeks. Eight years ago, he accidentally drove over a woman, breaking her leg ; was greatly alarmed at the occurrence, and felt his heart palpitate violently. Since that date palpitation has been readily provoked by excitement.

Six months since, his breathing became short, and he began to suffer from cough. Has never had hæmoptysis or œdema. Face and neck somewhat congested. Breathing embarrassed. Pulse 96, regular ; visible pulsation in carotid, temporal, and radial arteries. Cough, with frothy mucus expectoration. There is general emphysema of the lungs, with œdema of the bases of both, posteriorly. Precordial dulness, masked by emphysema of lungs. Apex-beat one inch outside nipple line, and in sixth intercostal space. Impulse diffused, but not strong. At the apex,

a loud, blowing, substitutive systolic murmur existed, audible likewise in the axilla and in the back; second sound faint. At the base there was a double murmur, the diastolic being the louder, blowing, and substitutive. Both were audible in the ascending aorta, and the systolic likewise in the arteries of the neck. Midway between apex and base no murmur was distinctly audible. R. Blister to front of chest, dry cupping to back, and a diuretic mixture.

18th. Much relieved as to breathing. Discharged.

Re-admitted on the 21st of March. At this date there was general œdema. No further note of the case has been preserved.

The most noteworthy circumstance in connexion with this case is the coincidence of the first symptoms of cardiac disease with the occurrence of nervous shock. A heart and aorta already in process of tissue deterioration, from the excessive and long continued use of alcoholic stimulants, as no doubt was the case here, and barely capable of performing their respective functions under the most favourable circumstances, cannot, without permanent injury, sustain a sudden revulsion from great emotional excitement or physical effort. Thenceforward, the symptoms of failing circulation will be more and more distinctly manifested; whilst tissue degeneration will make more rapid progress.

Further evidence of the truth of this proposition is supplied by the following case.

CASE XCIII.—*Angina ; Severe Mental Shock, followed by Aggravation of Angina, and Edema of Feet ; Pallor and Extreme Weakness ; Displacement of Apex-Point outwards ; Musical Systolic Murmur at Base, and traceable through Arch of Aorta into Arteries of Neck and downwards to Apex, likewise audible in Left Axilla and Back ; Second Sound Clear. Diagnosis : Aortic Obstruction by Calcareous Formation at Root and on Ventricular Aspect of Aortic Valve, Central and Marginal portions being Sound ; No Reflux ; Left Ventricular Hypertrophy ; No Mitral Disease.*

Eliza M., aged fifty years, a ladies' nurse, was admitted into the Mater Misericordiæ Hospital on the 23rd of April, 1870. Up

to two years prior to that date her health had been good, with the exception that, for some time previous, she had occasionally experienced dyspnoea, with pain shooting down the left arm after great exertion. She then received the startling news of the sudden death of her husband; the dyspnoea and pain in the arm became more frequent and more severe, and her feet soon became swollen. She never swooned, and never spat blood.

At the date of admittance, she was pallid. The radial pulse was not registerable, owing to extreme weakness; it was not visible. Feet slightly swollen. No engorgement of liver. No cough. Breathing in the recumbent posture tranquil. Precordial dulness not noticeably extended, but apex-pulsation half an inch external to the nipple-line. Impulse very feeble. At the apex, a loud, musical, substitutive, systolic murmur was heard; it was likewise audible in the left axilla, and at the inferior angle of the left scapula. Here, the second sound was sharp and clear, but weak. At midsternum a musical systolic murmur was likewise to be heard, and of a character and intensity which indicated this as its point of origin. From midsternum it was traceable upwards, in the course of the aorta, to the left clavicle, and also into the great arteries of the neck. It was likewise traceable downwards to the seat of apex-pulsation, losing force gradually, but undergoing no change of quality, as followed in this direction, or in that previously indicated.

At midsternum the second sound was sharp and clear, but faint; it was not accentuated in the pulmonary artery. The interval between the cardiac impulse and murmur on the one hand, and the radial pulse on the other, was not unusually prolonged. To have carbonate of ammonia, wine, and liquid food.

Two circumstances in connexion with this case are worthy of special notice; namely, that a murmur of aortic efflux in the adult was distinctly audible at the apex, in the left axilla, and at the angle of the left scapula; and that advanced disorganization existed at the orifice of the aorta, unaccompanied by inadequacy of the valves.

CASE XCIV.—*Acute Rheumatism; Basic Diastolic Bellows-Murmur, which subsequently became Harsh; Displacement of Apex Outwards. Diagnosis: Inadequacy of the Aortic Valves by Roughness of their Edges; Left Ventricular Hypertrophy.*

John W., aged twelve years, was admitted into hospital, May 12th, 1870, suffering from acute rheumatism of three weeks' duration, and engaging chiefly the joints of the right arm. He was pale, and suffering acute pain. There was visible pulsation of the carotids, but not of the radial or temporal arteries. The apex of the heart pulsated in the nipple line, and here a transmitted diastolic murmur was heard. A loud diastolic bellows-murmur existed at the base, and was transmitted to the apex, but not into the carotid arteries.

Four days subsequently the basic murmur was rasping in quality. Under treatment with colchicum and bromide of potassium, the affected joints being at the same time wrapped in cotton wadding, the boy soon got well of his articular symptoms, and abruptly left hospital without permission.

Judging from the point of apex-pulsation, I am clearly of opinion that the cardiac affection in this case was of, at least, several months' duration, and was probably due to the previous attack of rheumatism, of which, however, no history was obtained.

CASE XCV.—*Exposure and Intemperance; Oedema, Jaundice, and Hæmoptysis; Paroxysmal Dyspnoea of an Ascending and Descending Scale; Pulse Regular and Visible; Superficial Arteries Tortuous; Extension of Precordial Dulness; Apex-Beat in Nipple Line; Diffused and Heaving Impulse; both Sounds of Heart Masked at Apex; Postsystolic-Murmur at Base, and transmitted into the Carotids; Marked benefit from the use of Chloral; Repeated Puncturing, and Copious Discharge of Serum; Gangrene in Seat of Punctures; Death by Asthenia. Autopsy: Effusion into Pericardium; Enlargement and Granular Degeneration of Heart; Hypertrophy with Dilatation of Left Ventricle, and Thinning at the Apex; Dilatation, Atheroma, and Hyperæmia of the Aorta; Obstruction at the Aortic Orifice by Thickening of one of the Valve-Segments.*

Laurence C., aged thirty-eight, a gasfitter, and addicted to

tippling, was admitted into the Mater Misericordiæ Hospital, June 2nd, 1870. Has been much exposed to wet and cold in his avocation. Eleven weeks prior to date of admittance, his breathing became short, and three weeks later his feet began to swell.

When admitted he was suffering from orthopnœa, and likewise from paroxysmal dyspnœa, which exhibited the following peculiarities. Whilst he sat propped up in bed, there were alternations of suspended and of quickened respiration, passing into one another by gradations. The apnœal period, as timed by my watch, lasted twenty seconds, and during its continuance he was in a state of composure, and seemed free from suffering; then, there was a shallow inspiration, which was followed by an expiration of proportionate depth, and this was succeeded by several respiratory acts in increasingly rapid succession, till a rate of one in three seconds was attained, after which the rate gradually subsided to complete apnœa. The ascending scale extended over a period of thirty-six seconds, and in the progressive ratio of two, three, and four respirations in the successive periods of twelve seconds; the descending scale followed in the inverse order, and was of equal length; and finally, the apnœal period of twenty seconds completed the series.

During the period of ascent, the man moaned and tossed about for breath; he then experienced gradual relief till respiration was entirely suspended, when, as already stated, his tranquillity was complete. These variations of respiration were not attended with any alteration in the rate, force, or rhythm of the heart. Pulse 87, regular, small. Arteries in forearm tortuous, and pulsating visibly. Throbbing of carotids. Great œdema and rigidity of feet, legs, and penis. Conjunctivæ icteric. Urine 1·020 in sp. gr., and albuminous. Precordial dulness much extended; apex-beat diffused, and in nipple line; impulse diffused and heaving, but not strong. Both sounds at apex were somewhat masked. At the base, and in the course of the ascending aorta, and loudest rather above the site of the aortic valves, a soft, prolonged, postsystolic murmur was heard; it was likewise transmitted into the cervical arteries. Second sound not clear, but free from murmur.

6th. Spat some blood this morning for the first time. There has been a copious discharge of serum from punctures made in the penis. He has not slept since admittance, except on the night of the 4th, when he had a draught of grs. xv of chloral hydrate, and slept an hour and a-half. Hoffman's anodyne in 3ss doses failed to give him relief. Night draught of chloral (grs. xx) to be repeated. Chest to be dry-cupped, and one leech to be applied to the precordium.

On the evening of the 13th, he had an epileptiform fit, which lasted about an hour, during which he was unconscious and violently agitated. Subsequently, the thighs became enormously swollen; and from the tension so caused, great pain was experienced at the flexure of the groins. From punctures made in the groins and in the prepuce, there was a profuse discharge of serum.

Citrate of iron and quinine in gr. v doses was given thrice daily, and grs. xx of chloral hydrate at night. Through the agency of the latter he slept soundly every night except two, and on these two nights the chloral had been omitted by an oversight. On the 19th, the swelling of the thighs and legs had begun to subside, and respiration had become tranquil and regular.

28th. Chloral has been continued in similar doses, with the effect of insuring sound and refreshing sleep throughout the night. He usually awakes about 3 o'clock, A.M., remaining awake for about half an hour, and again sleeping till 9.30. Swelling less; breathing tranquil; appetite good. He is very cheerful and hopeful.

In the early part of July the thighs were again punctured; and although he continued free from suffering, and slept well at night under the influence of chloral given in the previous doses, he began to sink. Gangrene set in around the punctures in the thighs, and he died quietly on the evening of July 10th. Permission was granted by the relatives of deceased to examine the heart only. The pericardium contained about eight ounces of amber-coloured serum. The heart was globular, broad at the apex, and weighed twenty-two and a-half ounces. There were two well marked milk spots, one on the anterior, and the other on the posterior

surface of the right ventricle. The former was remarkably opaque, and consisted of hypertrophy of the sub-epicardial connective tissue, by which the serous membrane was sharply raised. The aorta was dilated above the sinuses, admitting three fingers; it was slightly atheromatous, and of a pink red tint on the internal surface. The aortic valves were competent and sound, with the following exception; viz., the right half of the right anterior segment was sharply thickened at the attached border; the thickened portion was smooth on the surface, and projected both on the arterial and ventricular aspects of the valve, less, however, on the ventricular surface, where it was invested by the lining membrane, but still projecting sufficiently to oppose an obstacle to the outflow of blood from the ventricle. The right chambers were normal, except that the anterior surface of the ventricle presented a thin layer of fat. The left ventricle was greatly dilated, thickened to three-fourths of an inch at the base and the middle, but thinned to two lines at the apex. The mitral valves were unaltered. The substance of the heart was somewhat granular. All four chambers contained yellow thrombi; those in the ventricles extended into the aorta and the pulmonary artery. The latter vessel was normal. Both coronary arteries commenced in one of the aortic sinuses.

CASE XCVI.—*Orthopnoea and Cough; Visible Radial Pulse; Weak Cardiac Action; Systolic Murmur at the Apex, and Double Murmur at the Base of the Heart; Systolic Murmur in the Arteries of the Neck; Death. Autopsy: Enlargement of the Heart; Dilated Hypertrophy of the Left Ventricle; Complete Disorganization of the Aortic Valves, and Thickening of the Aorta; Hypertrophy of the Coronary Plexuses.*

Barry W., aged thirty-eight years, a labourer, was admitted, July 23rd, 1870. Had drunk hard, but had never been affected with rheumatism. His breathing had been short for the preceding two years, but he had never spat blood. When admitted, he was remarkably sallow. There was orthopnoea, cough, and mucous expectoration, but no oedema. Pulse 84, soft, and visible. Car-

diac action weak; no impulse to be felt, and no precordial dullness, owing to the existence of pulmonary emphysema.

At the apex point, as localized by auscultation, a soft systolic murmur was heard, and at the xiphoid cartilage, both a coarse systolic and a soft postdiastolic murmur existed; the former was likewise audible in the carotid arteries. To have citrate of iron and quinine, and, at night, grs. xx of chloral hydrate. He became gradually weaker, and died on the 16th of August.

The heart weighed thirteen ounces. There was complete thrombosis of the right auricle, whilst the other chambers contained flakes of fibrin. The left ventricle was in a state of eccentric hypertrophy. The right and left segments of the aortic valve were rolled up and thickened, and the former was rough and calcareous. The posterior segment was somewhat thickened and shrivelled, but less altered than the other segments. The orifice was reduced to an irregular chink, and was permanently patulous. The root of the aorta was thickened, but not atheromatous. All the other orifices and valves were free from disease. The coronary plexuses were thickened, and the coronary arteries were dilated, but soft and pliant.

Remarks: There was a noticeable difference in quality between the systolic murmur at the apex, and that heard at the base of the heart. I cannot offer an explanation of this difference, except by assuming the occurrence of a dynamic murmur at the mitral opening.

The second sound heard to precede the diastolic murmur at the base, was that of the pulmonary artery; the murmur which represented the aortic second sound being posterior in time, owing to the dilated state of the left ventricle and aorta. In this respect, the case illustrates the remarks made at p. 126, in regard to the relative position in time, of the aortic and the pulmonary elements of a reduplicated second sound, in connexion with inadequacy of the aortic valves.*

* See also Case 99, last paragraph, p. 887.

CASE XCVII.—*Dyspnœa, Albuminuria, and Œdema; Visible Pulsation of Radial and Ulnar Arteries; Extended Precordial Dulness; Feeble Cardiac Impulse; Systolic Murmur at Base of Heart, and likewise in Ascending Aorta, Arteries of Neck, and at Apex. Diagnosis: Hypertrophy of Left Ventricle; Obstruction at the Aortic Orifice, and Atheroma of the Aorta and Arteries generally; Renal Disease. Marked Improvement; Discharge.*

John F., a waiter, aged forty years, was admitted, November 26th, 1870. Never had rheumatism, and had not been subject to fainting fits or dizziness. During the preceding two years he had noticed that his breathing became short when he was hurried. Three weeks prior to admittance his feet became swollen, then the genitals, and finally the hands. Urine 1·010 in sp. gr., contained albumen, and deposited lithates. Examined microscopically, it exhibited tube-casts denuded of epithelium, and large crystals of lithic acid. There was œdema of the feet, legs, and external genitals. Pulse 72, regular, and abrupt. Radial and ulnar arteries tortuous, and pulsating visibly on both sides. Breathing tranquil. Precordial dulness extended transversely. No distinct cardiac impulse to be felt, but a sensation of feeble heaving was communicated to the hand over the precordium generally. A harsh systolic murmur existed at the base of the heart and in the ascending aorta; it was heard in the arteria innominata, and likewise, but less distinctly, in the carotids, and at the apex of the heart. The second sound, wherever audible, was sharp and clear. R. Tinct. ferri, perchlor. ʒij; Æther. chloric, tinct. nucis vomicæ, a. a. ʒij; Infusi quassia, q. s. ad. ʒviiij. An ounce to be taken thrice daily.

December 9th. Urine again tested. Sp. gr. 1·020, and still containing albumen. Genitals repeatedly punctured since last report. Copious diuresis set in on the 7th, and continued up to the 9th. At latter date, the quantity passed in twenty-four hours amounted to six pints. A decided improvement has taken place in his general condition, and the œdema has nearly disappeared from the lower limbs and genitals.

15th. Left hospital to resume his work. Edema quite gone, but cardiac and arterial signs as previously reported.

CASE XCVIII.*—*Intemperance ; Fall upon the Chest ; Dyspnoea and Anasarca ; Visible Pulsation of Superficial Arteries ; Displacement of Apex Outwards ; Double Impulse ; a Systolic and a Prediastolic Murmur at the Right Base ; Sudden Death. Autopsy : Hepatic and Pulmonary Congestion, and Opacity of Capsule of Spleen ; Enlargement of Heart ; Dilated Hypertrophy of Left Ventricle, and Yellow Mottling of its Interior ; Incompetence of Aortic Valve by Relaxation and Perforation of Right Anterior Segment ; Dilatation and Atheroma of the Aorta ; Granular Degeneration of Discoloured Portion of Papillary Muscles.*

— D., a metal founder, aged thirty-four years, and formerly very intemperate, was admitted in September, 1872, under Dr. Nixon, who kindly invited me to examine him.

He had not had rheumatism ; but about two years prior to admittance he fell upon his chest, and sustained injury of an indefinite character, from which he subsequently experienced some slight inconvenience.

Two months prior to the date of admittance his feet became swollen, and his breathing short and oppressed.

At the date of my examination of him, he was very weak. Visible pulsation of radial, carotid, and temporal arteries ; radial pulse feeble, but regular. Apex-pulsation an inch and a-half to the left of the nipple-line. Impulse heaving, rather weak, and double ; the diastolic being the stronger of the two strokes, and accompanied by a faint dull sound. Immediately preceding the diastolic impulse and the accompanying (second) sound, a prolonged bellows-murmur, of medium loudness, was heard at the apex and in the left axilla. The first sound was faint and dull at the apex. At the base, two murmurs were heard ; viz., a faint, dull, systolic murmur, traceable through the aorta and into the neck, and a remarkably loud prediastolic murmur of a blowing character, which was audible throughout the arch of the aorta,

* This is the case referred to at p. 220, fourth paragraph.

and over the front of the chest generally. It was loudest at mid-sternum, and became gradually less distinct towards the apex. I had commenced my examination at the apex, and my first impression was that this murmur was presystolic, a mistake into which the dull quality of the succeeding sound, and the strong succeeding impulse led me, but which the soft blowing character of the murmur should have enabled me to avoid. On applying the stethoscope over the base of the heart, however, I at once identified the murmur as prediastolic in rhythm. Dr. Nixon was of opinion, in which, however, I could not concur, that a systolic murmur likewise existed at the apex.

The man died quite suddenly a few days afterwards; and on examination of the body, the liver was found to be congested, and the capsule of the spleen thick and opaque. The bases of both lungs were congested. The heart was enlarged, and weighed eighteen and a-half ounces. The right cavities were normal, and contained some clot. The left ventricle was greatly dilated, and its walls were somewhat thickened at all points except the apex, and here the thickness of the wall did not exceed three lines. The interior of the ventricle was somewhat opaque, and mottled yellow. The mitral opening was somewhat dilated, but not disproportionately to the ventricle, and the mitral valve was correspondingly expanded, and competent to close the passage. There was slight thickening of the anterior flap near the edge, but only by extension of the attached and thickened tendinous chord. The left auricle was normal. The aortic valves were atheromatous, especially the right anterior segment, which, when the valve was closed down, hung loosely into the ventricle several lines below the level of the other two segments, leaving a passage as large as the tip of the index finger, by which water poured into the aorta flowed at once into the ventricle. In the centre of the right segment of the valve there was, moreover, an opening as large as a pea, with jagged edges, and near this, another of the diameter of a pin's head. The aorta was dilated, and internally it was somewhat red in patches, and rough by atheromatous change.

Doctor Nixon reports that the discoloured portion of the papillary muscles, examined microscopically, was found to be in the granular stage of fatty degeneration.

CASE XCIX.—*Intemperance ; Strain of Chest ; Œdema, Orthopnoea, and Paroxysmal Dyspnoea ; Visible Pulsation of Superficial Arteries ; Congestion of Liver and Lungs ; Extension of Precordial Dulness ; Strong and Double Impulse of Heart, and Displacement of Apex Downwards, and to the Left ; Systolic Murmur at Apex, and in Left Axilla and Back ; Systolic and Postdiastolic Murmur at Right Base ; Sudden Death. Autopsy : General Enlargement of the Heart ; Dilatation and Thinning of the Right Ventricle ; Dilatation and Thickening of the Left Ventricle ; Slight Thickening of the Mitral Valve ; Softening and Laceration of Two Segments of the Aortic Valve ; Atheroma and Dilatation of the Aorta above the Valves.*

Michael D., aged forty-five years, a brewer's carter, who had drunk freely of porter, was admitted into the Mater Misericordiæ Hospital in October, 1872. Nine weeks previously, whilst engaged with other men in lifting a cask of porter up a ladder, he overbalanced himself, and would have fallen to the ground had he not held on by one of the rungs of the ladder, from which he suspended himself. He felt a strain in the chest for a moment, but soon recovered, and was able to resume his work. Six weeks later his feet began to swell.

When admitted, he was suffering from orthopnoea, with occasional paroxysms of dyspnoea. Respiration 36. Feet and legs much swollen. Pulse 84, regular, full and strong, but visible. Precordial dulness was extended horizontally, and the impulse of the heart was heaving, strong, prolonged, and double. The apex-beat was felt in the sixth intercostal space and nipple line. A soft systolic murmur was audible at the apex, and in the left axilla and back. At midsternum a double murmur was heard ; viz., a faint, somewhat harsh systolic, and a soft postdiastolic murmur, the second sound being sharp and clear. The former of these murmurs alone was audible in the arteries of the neck. Urine 1·015, acid, depositing lithates on cooling, and containing a trace of albumen. Liver enlarged and congested. Lungs congested.

Death took place rather suddenly. On examination of the body, the heart was found enlarged, weighing nineteen ounces,

globular in figure, and exhibiting two large "milk spots" on the right ventricle; viz., one on its anterior, the other on its inferior surface. The pericardium over these spots was smooth and glistening. The right ventricle was dilated and thinned, and both it and the right auricle contained some dark coagulum. The left ventricle was dilated, and its wall at the base and middle was three-fourths of an inch thick. The mitral valve was competent, but slightly thickened at the free margin. The right and left anterior segments of the aortic valve were thickened, brittle, and rent across the centre, the free edges not being engaged, but forming "bridles" across the orifice when the valves were shut down. When the valves were closed, the rents in them readily admitted the point of the little finger. The aorta was atheromatous, and much dilated above the level of the valves.

There is good reason to conclude that the aortic valves, previously to the occurrence of laceration, had been both obstructive and inadequate, and that then, as subsequently, the second sound was double; a murmur of reflux, which was postdiastolic in rhythm, owing to the dilatation of the left ventricle, representing the second sound in the aorta. The second sound of the pulmonary artery was audible at the right base, and preceded the aortic murmur.* It was, no doubt, owing to the previous existence of regurgitation at the aortic orifice, that accidental laceration of the valves, nine weeks prior to admittance, failed to develop new symptoms of cardiac and respiratory distress. The dependence of paroxysmal dyspnoea on atheromatous change and dilatation of the aorta, and of diastolic impulse upon excentric hypertrophy of the left ventricle, are well exemplified in this case.

CASE C.—Hæmoptysis; Displacement of the Apex of the Heart Outwards; Double Basic Murmur; Sudden Occurrence of Right Hemiplegia, with Aphasia and Amnesia; Rapid Recovery of the Use of the Arm and Leg, and of the Organs of Speech; Imperfect Recovery of the Remembrance of Words. Diagnosis: Capillary Embolism of the Left Hemisphere of the Brain.

James M., aged forty years, a corn porter, admitted, November

* See p. 882, last paragraph.

the 8th, 1872. Never had rheumatism, but spat blood occasionally. Was accustomed to push heavy loads with his arms. Complained of great weakness. Pulse 84, full. Apex-beat in nipple line, and felt over an area of an inch and a-half in diameter, and here two transmitted murmurs were heard. At base two murmurs were likewise audible, of which that of systolic rhythm alone was transmitted into the carotid arteries, and was audible in the left back. The second sound was not intensified in the pulmonary artery.

On the evening of the 20th he was in his usual state, and in bed, having apparently undergone slight improvement in regard to sleep and feeling of weakness. On the following morning he was "queer," and when I saw him at 11 A.M., he answered questions incoherently and with difficulty; was feverish, and the right eye was hyperæmic. I expressed to the class my suspicion that capillary embolism of the brain was taking place.

On the 22nd he was hemiplegic on the right side, scarcely able to utter a word, and quite "astray."

On the 23rd he had, in some degree, recovered the use of the arm and leg, and was more intelligent; the left eye was now more injected than the right.

On the 24th he had recovered the perfect use of his limbs; he answered promptly but irrelevantly, and both eyes were injected.

25th. He was quite intelligent, but had forgotten the proper names of things. When, however, the correct names were mentioned, he at once, and with apparent satisfaction, recognised them, and repeated them without difficulty. When asked the name of an object which he did not remember, he tried hard to recall it, and frowned in the effort to do so. I asked him to name the day of the week (Monday) on which he was admitted; he answered "Thursday," but with apparent doubt as to his accuracy. I then asked him was it not "Monday," and he immediately, and with evident satisfaction, answered, "Yes, Monday." When I placed my finger on his great toe, and asked him what it was, he said "Thursday;" but when I inquired was it not his toe, he answered, with a smile, "Yes, toe."

After a course of treatment with iodide of potassium and

infusion of cinchona, he was discharged on the 3rd December, having quite recovered the use of his limbs and the faculty of speech. His remembrance of the names of things was, however, still imperfect, and the cardiac signs remained unaltered.

CASE CI.—*History of Rheumatism and Severe Pain in Right Side ; Recurrent Angina Pectoris ; Orthopnœa ; Œdema, Cough, and Hæmoptysis ; Feeble Cardiac Impulse and Faint First Sound ; Accompanying Diastolic Murmur at Base ; Death. Autopsy : Effusion into all the Serous Cavities of the Chest ; Heart Enlarged and Fatty on Surface ; Thrombosis of Right Chambers ; Hypertrophy and Dilatation of both Ventricles ; Great Dilatation of Tricuspid Opening ; Valves all Structurally Sound, but Aortic Valves Inadequate by Dilatation of the Aorta, which was likewise Atheromatous ; Osteoid Deposit between Base of Right Lung and Diaphragm.*

John F., aged fifty years, had served twenty-one years in the Metropolitan Police Force; became an extern patient of the Mater Misericordiæ Hospital in the early part of 1866. He was then suffering from dyspnœa, with slight cough and expectoration. I soon lost sight of him; but subsequently learned that in May, 1867, he had an attack of rheumatism, which was accompanied with severe lancinating pain in the lower part of the right side. I next saw him in February, 1868. The feet and legs were then swollen; dyspœna had become much aggravated, and he coughed incessantly.

On the 20th of March following he called at my house in a state of the most extreme respiratory distress, which required for its alleviation the free use of wine and ether, and the admission of air by the window. I now learned that for the previous four weeks he had not been able to lie down, such was the difficulty of breathing in the recumbent posture. He was admitted into hospital on the 21st of March, and at that date his condition was as follows: The feet, legs, and thighs were greatly swollen and tense, but not erythematous. Urine 1·018 in sp. gr., acid, and, on cooling, exhibited a brick-dust deposit which disappeared on the application of heat. There was venous and arterial throb-

bing in the neck, and the supra-clavicular fossæ were obliterated. The area of precordial dulness was much extended, the impulse of the heart was feeble, and over the apex the first sound was somewhat masked and ill pronounced, but free from murmur; whilst the second sound was accompanied by a soft and faint murmur. At midsternum, the first sound was masked and prolonged, but without murmur, whilst the second sound was obscure, and attended with murmur of a soft and blowing character, extending upwards and to the right with scarcely diminished intensity. There was congestion of the bases of both lungs, and respiration was universally feeble, and accompanied with râles. Under treatment, consisting of stimulants and anti-spasmodics, the man improved considerably, so that he was soon able to lie down, and even to sleep in the recumbent posture.

His condition remained nearly the same from the 30th of March to the 4th of April. During this interval, Dr. Grimshaw kindly took the subjoined tracings of the pulse (Figs. LIII. and LIV.) which, as will be seen, are characteristic of hypertrophy with dilatation of the left ventricle, and aortic regurgitation without obstruction. They were in all particulars confirmatory of the diagnosis previously made.

FIG. LIII.

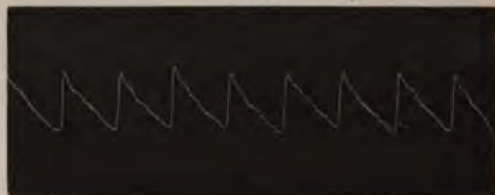
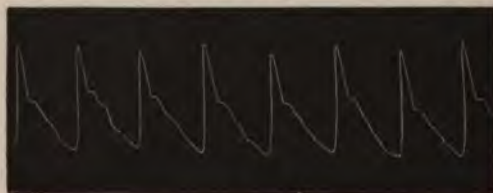


FIG. LIV.



On the 2nd of April there was, for the first time, hæmoptysis;

and this continued without intermission till his death, which took place at 11 P.M. on the 4th of April.

The body was examined fifteen hours after death. There had been very considerable effusion into both pleuræ, and into the pericardium. The left lung was emphysematous, and along the anterior margins of both there had been considerable extravasation of blood. The base of the left lung was likewise, but to a less extent, the seat of extravasation.

The heart was greatly enlarged, fatty on the surface, and weighed twenty-five and a-half ounces. The right chambers were occupied by a large mass of partially decolorized fibrin, which likewise passed for some distance into the pulmonary artery. The right ventricle was somewhat dilated and thickened, and the tricuspid orifice readily admitted the four fingers and thumb. The left auricle had undergone no apparent change, but the left ventricle was greatly thickened and dilated. The mitral orifice was somewhat dilated, but the valves had undergone proportionate expansion, and were healthy and competent. The aortic orifice was greatly dilated, but the aortic valves were healthy, except that the attached margins were somewhat thickened; they were, however, incompetent, owing to the dilatation of the orifice. The aorta, for a distance of three inches above the orifice, was greatly dilated; it was likewise atheromatous, but not rough internally. The lining membrane was hyperæmic.

The liver was much enlarged; the spleen was normal; the kidneys were congested, the cortical structure was thickened, and there was a good deal of fat in the pelvis.

Between the base of the right lung and the upper surface of the diaphragm, there was found a plate of osteoid material, two or three lines thick and two and a-half inches in diameter. It was concavo-convex, and strictly adapted to the surface of the lung and the diaphragm, to both of which it was firmly attached. Microscopic examination of this plate showed that it possessed many of the histological characteristics of normal bone. Thus, it exhibited lacunæ, canaliculi, and large channels, not unlike the Haversian canals. They differed, however, in some degree, from the corresponding features of bone, in regard to size, figure, and regularity of arrangement. The annexed woodcut, from an

admirable sketch made with the camera by Dr. E. W. Collins, shows accurately the characteristic features of this adventitious structure.

FIG. LV.



In 1844, Dr. Law exhibited before the Pathological Society of Dublin, a specimen of pseudo-ossific deposit in false membrane attached to the pleura; and he mentioned to me another example which had come under his notice, of a similar deposit between the base of one of the lungs and the diaphragm.

Doctor Hillier brought under the notice of the Pathological Society of London,* in 1855, a case in which osteoid nodules were dispersed extensively through the soft tissues of the body. He found them in the lungs and in the diaphragm. The illustrations, annexed to his report, of the microscopic characters exhibited by sections of these nodules, resemble Fig. LV. in many respects. Dr. Hillier regarded the morbid formations in his case as cancerous, cancer having existed in other parts of the body. No appearance whatever, even remotely suggestive of cancer, was exhibited in my case.†

Diseases of the mitral, as those of the aortic valves, may be

* *Vide Reports*, vol. vi., page 317.

† For details of the preceding Case (101), see also *Proceedings of the Pathological Society of Dublin*, vol. iii., part iii., new series, page 277.

classified under the two heads of lesions of obstruction, and lesions of regurgitation.

Obstruction of the mitral orifice arises, in many instances, from primary inflammation and contraction of the auriculo-ventricular ring, and should be designated annular rather than valvular. In such cases, the disease has its origin in sub-acute inflammation, most frequently rheumatic, of the tendinous ring, engaging the valves secondarily and by extension. It is most frequently met with in children, and is characterized pathologically, in its ultimate stages, by extreme contraction of the orifice, thickening of its boundary, and cohesion and shrinking of the valves, constituting veritable cirrhosis. The aperture thus narrowed, whilst retaining its natural figure, and bounded by seamed, puckered, and well-defined edges, presents on the auricular aspect a very remarkable appearance, appropriately designated the "button-hole" form of contraction. It was exemplified in Cases 102 to 113 inclusive. In adults, and in persons of middle age, more especially those of a gouty diathesis, calcareous nodules are frequently formed upon the edges of the opening by consecutive degradation of tissue. Annular, as distinguished from valvular stenosis, is by many degrees more common at the mitral than at the aortic orifice. This difference depends, no doubt, upon the greater quantity of white fibrous tissue involved in the left auriculo-ventricular ring.

The button-hole contraction of the mitral valve is occasionally so modified as to present the appearance of a diaphragm, stretched horizontally in the plane of the opening, with a central aperture resembling a hole in a drum-head. This aperture frequently retains the elongate figure, and the direction, of the slit exhibited between the closed valves in the normal condition; the edges, however, being thick and tuberculated. On the auricular aspect the outline of the contracted slit is crescentic. The pathology of this modified narrowing is identical with that of the button-hole contraction, which it represents in an early stage. Its origin is usually rheumatic, and the subjects of it are most frequently children or adults.

The form of lesion designated "funnel-shaped" stenosis, is the result of primary acute inflammation and thickening of the valve

segments, with cohesion of their adjacent edges.* The obstruction thus produced leads to hypertrophy of the left auricle, and from this, in turn, arises increase of pressure upon the valves, which are forced downwards into the ventricle and assume the appearance of a funnel, with a more or less contracted orifice, projecting by its small extremity into that chamber. Cases 102 and 116 furnish examples of this form of contraction.

The relative numerical proportion in which mitral stenosis assumes these two forms respectively, has been variously estimated. Of the 15 cases in which I was enabled to determine this question positively by *post mortem* examination, 13 presented examples of the button-hole form of constriction, and 2 only that known as the funnel-shaped form. Of the former list, 8 were connected with a history of rheumatism; and in 4 of these, aortic was associated with mitral valve-disease. Of the 47 cases recorded by Dr. Hilton Fagge, the contracted mitral opening had assumed the "button-hole" form in 46 instances, and the funnel-shaped configuration in 1 only.† Since my attention was first directed to constriction of the mitral opening, ten years ago, I have had under observation, and have carefully noted, 81 examples of it. Of these, 63 are included in the principal list (Table XI.), upon which my statistics of the disease are based. The remaining 18 cases have been noted since that table was constructed, and are included in the supplementary list. (Table XII.)

The *symptoms* of mitral constriction, disassociated from the physical signs, are by no means characteristic. Most of them may be witnessed in other forms of valvular lesion. A few of these symptoms, however, are eminently suggestive of mitral obstruction, as distinguished from other forms of cardiac disease, and are present at an early period in cases of valvular disease, with rare exceptions, only where this lesion exists. This is especially true in regard to sharp pain occasionally felt at the apex of the heart, and hæmoptysis.‡ It has been stated, and

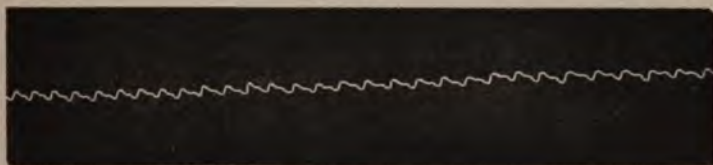
* See p. 801.

† *Guy's Hospital Reports*, series iii., vol. xii.

‡ I do not contemplate the possibility of confounding this with pseudo- or hysterical pain.

somewhat generally believed, that the pulse of mitral constriction is quick, small, weak, and irregular. In the advanced stages of the disease, when the left ventricle begins to fail, and congestion of the lungs, engorgement of the right chambers of the heart, general venous obstruction and anasarca have taken place, such is, no doubt, the character of the radial pulse (see Fig. LX.); but in these respects it differs not from the pulse of simple mitral regurgitation at a corresponding period of that disease. In the earlier stages, and up to the period when, in the ordinary course of the disease, the changes just mentioned have taken place, the pulse of mitral obstruction is usually quite regular, and not often above 90 in the minute, but small.* (See Figs. LXII, LXIII, and LXV.) It is, however, easily quickened by exercise or moderate exertion, and is then associated with palpitation, dyspnoea, and pain at the apex. In a few

FIG. LVI.

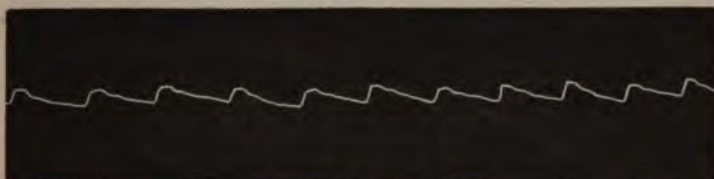


Mitral constriction (extreme).

Michael C. B. Pulse 114. September 22nd, 1873.

I have, unfortunately, lost my notes of this case; it is, therefore, not included in the Tables.

FIG. LVII.



Mitral constriction. Pulse 60.

Thomas D. (No. 39, Table XI.) Slight irregularity and inequality of pulse.

June 13th, 1873.

* Dr. Fagge says (*loco citat.*, p. 76), "It has appeared to me that in the great majority of cases in which a presystolic murmur has been heard, the pulse has presented no indications whatever of the existence of disease."

cases the pulse is permanently quick. The annexed tracing (Fig. LVI.) represents the only example of this kind which I have met with.

It may, on the other hand, be permanently slow, as shown in Figs. LVII. and LVIII.

Respiration is habitually short, and readily accelerated by physical effort of any kind, especially by that of making an ascent. There is generally short and teasing cough, with little or no expectoration, and a feeling of tightness in the chest. The temperature is low, the patient is easily chilled, very susceptible to cold, and liable to intercurrent attacks of bronchitis.

Occasional expectoration of pure and florid blood is a common symptom, whilst dropsical effusions are very rare; occurring only in the ultimate stages, when, by failure of the ventricles, pulmonary and general venous congestion has taken place. This remark applies also to congestion of the liver, spleen, and kidneys, accompanied with epigastric tension, and abundant deposit of lithates, with a trace of albumen in the urine. Death is, in most cases, directly caused by œdema of the lungs, which is attended with great difficulty of breathing, orthopnoea, and increased hæmoptysis, and by serous effusion into the pleuræ and pericardium.

FIG. LVIII.



Mitral constriction and regurgitation. Hypertrophy and dilatation of left ventricle. Albuminuria and hæmaturia. Michael O'B. (No. 18, Table XII.) Pulse 36. September, 1874.

The *physical signs* of mitral obstruction are, however, in the highest degree characteristic; indeed, carefully studied and identified, they are pathognomonic of the disease. These signs are two; namely, fremitus and murmur of presystolic rhythm. They have been so fully discussed in a previous chapter,* that a brief recapitulation of their salient features is alone necessary here.

* Chapter iii., p. 213.

The fremitus of mitral narrowing is located at the apex, and is strictly limited to the apex area. It communicates to the hand a jarring or vibratory sensation; it is usually, but not always, well pronounced; and it immediately precedes the systolic impulse of the heart.

Corvisart fully recognized the value of precordial fremitus as an element in the diagnosis of mitral narrowing. With reference to the signs indicative of this affection, he remarks: "In this number is a particular rustling, difficult to describe, perceptible to the hand when placed on the precordial region, and which, doubtless, proceeds from the difficulty which the blood experiences in passing through an aperture no longer proportionate to the quantity of fluid to which it has to give vent."*

'Apex fremitus, not due to mitral obstruction, is exceedingly rare. It may be caused, however, by mitral regurgitation, the valves being rough or spiculated, as well as inadequate; or by aneurism of the apex of the heart.

The fremitus of mitral reflux, besides being a very rare phenomenon, is strictly systolic in rhythm, and therefore *coincides* with the impulse and the first sound of the heart; whereas, that of mitral obstruction is distinctly presystolic, and *precedes* the impulse, as already stated. Fremitus, due to aneurism of either ventricle, is most frequently *diastolic*, and when not of that rhythm, it is always systolic.†

So characteristic and so easy of recognition is the fremitus of mitral obstruction, that from it alone, on applying my hand to the precordium, and before proceeding further with the examination of the patient, I have repeatedly and correctly diagnosed that lesion. It is not, however, a constant symptom. It is frequently absent where murmur announces, in the most positive manner, that mitral narrowing actually exists. Out of the total of 81 cases included in Tables XI. and XII., it was present only in 41 instances, or a little more than one-half of the whole number; and of the 15 cases in which death occurred, and the body was examined, it was noted in 6 only.

Presystolic murmur is, in the strictest sense, pathognomonic

* *The Diseases of the Heart*, Hebb's translation, p. 206.

† See Cases 69 and 70, pp. 759 and 763.

of this condition. It is *never* present where mitral narrowing does not exist, and it is *rarely* absent, and then only for a very limited period, in cases of that lesion.* Dr. Hilton Fagge failed to detect it in 4 cases, the patients being at the time *in articulo mortis*. He is, however, of opinion, that "it is yet to be proved that such an affection of the mitral valve can be present in any marked degree, and yet, while the heart is beating steadily and quietly, no presystolic murmur be at any time discoverable."†

In some recent lectures,‡ exhibiting great originality, but promulgating, as I venture to think, very unsound doctrine, Dr. Whyte Barclay has endeavoured to discredit the views generally held by physiologists in regard to the rhythm of ventricular systole, and to modify essentially the current pathology and the significance of presystolic murmur. He alleges that "the sounds of the heart do not indicate either the commencement or the termination of the systole;" that "the ventricles begin to contract before the long silence is concluded;" that "when the ventricle is full, the onward current through the mitral stops, then the systole begins, and before its termination the first sound occurs;" and finally, he urges that it is quite impossible the first sound and the impact on the "ribs can occur, until after contraction has proceeded to some extent." In reference to the last statement, I will only observe that it would imply that the pulsation of the aorta and of the carotid arteries should necessarily precede the first sound; such, however, is not the case.

He surmises that, "owing to some circumstance, the closure of the mitral valves may be postponed, whilst the other elements of the first sound are masked or muffled. Thus, the first sound is sharp, the short pause shortened, and the long pause lengthened, while, during the portion of systole preceding the valve-element, a murmur may occur, which yet may be systolic." But ventricular systole competent to educe valve-click, and of presystolic rhythm, should necessarily be accompanied by presystolic pulsation of the aorta and its primary branches. Hence, carotid pulsation should precede the first sound, which it never does, and presystolic murmur should be synchronous with the

* See Tables XI. and XII., and p. 901.

† *Loco citat.*, p. 73.

‡ *Lancet*, March 2nd and 16th, 1872.

carotid pulse, which it is not. He recognizes two distinct stages of ventricular systole; namely, "tension," and "shortening" of the fibres, "the tension of the fibres being complete when the impulse is felt, and the first sound occurring somewhere in the middle of the contraction, at the point, in fact, where tension is complete and shortening begins." Finally, he regards presystolic murmur as due to mitral regurgitation during the state of "tension" of the ventricle.

But ventricular tension, adequate to the development of a reflux murmur at the mitral orifice, should likewise cause efflux by the normal outlet, and pulsation of the carotid arteries; whilst its maximum force, exerted "at the middle of contraction," and causing the impulse and the first sound, should, in every such case, give rise to a second and louder murmur of systolic rhythm. Now, it is scarcely necessary to urge, that the true presystolic murmur does *not* coincide with carotid pulsation, but *precedes* it by a distinctly appreciable interval; and that mitral systolic murmur is rather an exceptional than an ordinary accompaniment of presystolic murmur.

Doctor Cryan has brought under the notice of the Pathological Society of Dublin, an example of mitral and tricuspid narrowing, in which presystolic murmur could not be detected, although the patient had been fifteen days under his observation in hospital, and during that time had been repeatedly examined by him.* From the general features of the case, notwithstanding the absence of presystolic murmur, the diagnosis of mitral narrowing was made. Dr. Cryan thinks that the absence of murmur may have been due to extreme contraction of the mitral orifice, which was, in this case, actually reduced to the diameter of the point of the little finger; the tricuspid opening being likewise greatly contracted. The diagnosis was very creditable to Dr. Cryan's acumen; but his explanation of the failure of murmur is to me not entirely satisfactory. I have had under observation examples of still greater narrowing of the mitral opening, in which presystolic murmur was well pronounced, and continued to be heard throughout.

* *Proceedings of the Pathological Society of Dublin*, vol. iv., part ii., new series, 1870.

Doctor O'Ferrall was of opinion that, in many instances, narrowing of the mitral orifice is consecutive to, and compensatory of, shortening of the mitral valves from previous endocarditis.* He adduces, in support of this opinion, three cases which came under his notice. In these cases there was systolic murmur, with fremitus, at the apex; the murmur ceased sometime before death, and the mitral orifice was found greatly contracted. He believed that, in endocarditis, the valves are affected before the orifice; hence regurgitation precedes obstruction. In proportion as the orifice is involved at a later stage, it is narrowed, and so adapted to the shortened valves; hence the cessation of a previously existing murmur. This ingenious theory is based upon a fact and a postulate. The segments of the mitral valve were found to overlap one another; hence "the mechanism of the parts seemed incompatible with regurgitation;" and a murmur of mitral constriction, if present, "should have been in connexion with the second sound." It is most probable regurgitation had not at any time existed in these cases; that the murmur actually present in the first instance was presystolic, a supposition certainly not excluded by the test of rhythm applied; and, that its suppression at a later period was due to the extreme debility which is admitted to have been present.

Doctor W. R. Sanders has found the presystolic murmur to be that which has varied most remarkably. He adds, that exertion on the part of the patient is generally, but not always, competent to educe it, where mitral narrowing exists.†

Doctor W. R. Gowers, on the other hand, maintains that the assumption of the erect posture has precisely the opposite effect upon this murmur. Drawing his conclusion from his experience of four cases, he asserts that the presystolic murmur is "louder and longer in the recumbent than in the erect posture," and that "in many instances it may be heard in the former, and not in the latter posture."‡ In reference to the effect of change of posture, my experience rather coincides with that of Dr. Sanders. In no instance have I found the murmur of mitral constriction less

* *Dublin Journal of Medical Science*, vol. xxxii., July, 1843.

† *Edinburgh Medical Journal*, vol. xiv., 1868.

‡ *Practitioner*, December, 1873.

loud in the erect, than in the sitting or recumbent posture. Indeed, in every case of the kind that has come under my notice the murmur in question has been intensified by the effort of standing up.

Doctor Waters believes that presystolic murmur is "far more frequently absent than present, even when there is great obstruction at the mitral orifice."* He gives five examples within his own experience in support of this opinion. The first of these patients was under observation nearly a month; the second, for several weeks; the third, a few hours; the fourth, two months, and subsequently for five days; and the fifth, nearly three weeks. In all these cases, mitral systolic murmur was heard, but presystolic murmur not at any time. The mitral orifice was contracted in each of these cases.

Where extreme debility from any cause exists, the murmur is most frequently suppressed in the agony of dissolution, and this may continue for several days.† It may be in abeyance for a longer or shorter period, according to circumstances. Thus, I have repeatedly failed to detect it on the first examination of a patient recently admitted, and still suffering from exhaustion due to want of proper food, and to the fatigue attendant upon removal to hospital. Even in such cases, however, suspicion as to the nature of the case having been aroused by the preceding and attendant symptoms, an ill developed murmur may, by strained attention, be detected in the presystole. From this, in conjunction with other evidence, I have ventured, in several instances, to make the presumptive diagnosis of mitral obstruction, and have found my diagnosis confirmed by well pronounced murmur on the following day. In 2 cases only, out of the 66 not fatal, has a presystolic murmur, previously identified, ceased to be audible whilst the patient was under observation. After two days, however, it returned in 1 of these cases, and continued to be heard during several weeks, till she was discharged. Three years subsequently, this patient was again received into hospital, with the signs of mitral narrowing still more distinctly pronounced. In the second case, the cessation of murmur coincided with great

* *Diseases of the Chest*, second edition, 1878.

† See Case 108.

weakness and irregularity of pulse, and was again audible on the following day.*

Of the 15 fatal cases in which mitral contraction was proved by autopsy to have existed, there were only 3 in which presystolic murmur was not at any time heard. In one of these cases the patient was nine days under observation previous to her death. She was, however, hemiplegic and aphasic, and the heart acted with extreme feebleness and irregularity during the whole of that period.†

The second case was under observation five days, and, although in a very weak state, the patient was not moribund during the greater portion of that time. I examined her daily, and I am convinced presystolic murmur cannot have then existed. Its absence, as already stated (p. 712), I am disposed to attribute to enfeeblement of the left auricle, by the pressure of a copious effusion into the pericardium and pleuræ.‡

The third case afforded the most remarkable example of suppressed presystolic murmur which has come under my notice. I use the word "suppressed" advisedly, because, in view of the condition of the mitral orifice discovered after death, I concluded that murmur of this rhythm must, of necessity, have existed at an earlier period of the man's illness. He had been thirty-nine days under observation, had been repeatedly examined, and the *seat* of the murmur heard had been frequently discussed with my colleague, Dr. Nixon, and with the class; but its *rhythm*, which was by all of us regarded as systolic, had never been called in question. I am satisfied, therefore, that presystolic murmur had not been pronounced at any time during the man's residence in hospital. I can offer no explanation of the suppression in this, as in the cases previously referred to, except the debility of the heart.§

In Case 68 (p. 749) presystolic murmur was inaudible when the patient was first examined, neither was it heard for several days after my second visit to her. It was then detected, and

* See Cases Nos. 8 and 9 in Table XII.

† See Case 103.

‡ See Case 61, p. 710.

§ See Case 114.

continued to be distinctly audible from that date till a short time before her death. The body was not examined.*

In the case of the woman, Jane McG. (109), presystolic murmur was not detected till eighteen days after her admittance. During this period the pulse was very weak, and the heart was repeatedly examined. For some time preceding death, the action of the heart was not equable, and murmur was heard only during the strong contractions of the ventricle.†

Thus, in 2 cases only out of a total of 81, or in the proportion of 2·4 per cent. was the murmur actually suspended under my personal observation, yet in both of these it was resumed after a brief interval. In the first, I cannot explain the suspension; but in the second, great weakness of the heart existed.

Amongst the fatal cases, presystolic murmur was not at any time heard in 3 instances out of 15, or in the proportion of 20 per cent.; and in a third case, it was detected only after the patient had been eighteen days under observation. In a fourth case, not included in this list, because the body was not examined after death, murmur was detected only a week after the patient had come under my notice.

In the case of the girl Eliza B. (No. 12 in Table XII.), presystolic murmur was developed during a secondary attack of rheumatism. I conclude that in this case the mitral orifice had been previously narrowed, but not in a degree adequate to the production of murmur.

In every instance in which I have heard a veritable presystolic murmur, I have confidently diagnosed narrowing of the mitral or tricuspid orifice, according to the seat of the murmur; and of both orifices, where the locality of murmur was dual. Yet, in no single instance has the diagnosis so made been falsified by the event.

Judging strictly within the limits of my own experience, and drawing my conclusions from the statistics above given, I feel justified, therefore, in holding,

(a) That presystolic murmur is pathognomonic of auriculo-ventricular narrowing;

* See Case No. 14 in Table XII.

† See Case 109, and No. 7 in Table XI.

- (b) That it is never present where auriculo-ventricular narrowing does not exist ;
- (c) That it is never permanently absent in cases of this lesion ;
- (d) That it is very seldom temporarily absent in such cases ;
- (e) That in these exceptional instances, the explanation of its absence is almost invariably to be found in debility of the heart ;
- (f) That the form of cardiac debility with which the suspension of presystolic murmur is most frequently associated, is that of the process of dissolution ;
- (g) And, finally, that the duration of suspension depends upon that of cardiac debility, and *in articulo mortis* it may extend over several days ;

Presystolic murmur may exhibit four different varieties of rhythm, as already stated.* I have, in more than one instance, observed these several varieties to shade off into one another, in the order in which they are set down, concurrently with the progress of the disease. The second form has been discussed at p. 206, and also in connexion with an illustrative case at p. 697.† The third form is exemplified in Case 117.‡ The prolonged murmur may undergo an ulterior change of abbreviation, by suppression of its terminal and intermediate portions, the postdiastolic portion alone remaining, as exemplified in Case 116. Even an alternate fusion and resolution of the postdiastolic and presystolic elements of this compound murmur may be observed to take place, in periods of alternating excitement and composure of the heart. Case 117, affords an example of this kind. The fourth form indicates a rough state of the constricted mitral orifice, and great debility of the left auricle ; a condition, in short, proximate to that with which extinction of murmur is associated. It is exemplified in Case 113.

Doctor George Balfour is virtually in agreement with me in

* See p. 214.

† The cases mentioned at p. 214 as illustrative of this variety of the murmur indicative of mitral narrowing, are Case 116 in the text, and Nos. 39 and 48 in Table XI.

‡ See also Nos. 15, 39, and 42, Table XI.

reference to the variation of form three, or rather he has anticipated me in the view just stated.* He says, in effect, that the murmur (presystolic) often follows the second sound immediately, being separated by an interval from the presystole, but sometimes running through to the apex-beat. These two forms are, in his opinion, indicative of great contraction of the orifice. The constriction, as he truly remarks, is usually of the diaphragmatic kind; but I doubt he is equally correct in holding that prolonged murmur and sharp first sound indicate a funnel-shaped condition of the mitral opening.

Doctor Whyte Barclay has recorded a case in which a protracted diastolic pause was entirely covered by a double murmur; the first portion faint, soft, and commencing immediately after the second sound; the second loud, harsh, and ending in the first sound.† This was, in my view, an example of the modified form of presystolic murmur which I have described in category number two.‡

Taken alone, and in its typical form, the presystolic murmur of mitral stenosis may be represented by the series *r-r-r*; and in combination with the first sound, which is frequently clear and abrupt, it may be mimicked phonetically by the formula *r-r-r-up*, sharp. The postdiastolic element, whether alone or combined, is, however, usually of soft and blowing quality. Indeed, I can recall only one case (116) in which it was harsh. It presented this character for a brief period only, and was then accompanied with fremitus. Presystolic murmur is strictly confined to the area of the apex in the great majority of cases; and when audible beyond this boundary, it is so only to a very limited extent, very faintly, and manifestly by transmission. I have heard it in the back in one case only (Case 106); in the axilla, in three cases (Cases 106, 117, and No. 12, Table XI.); and I have seldom heard it more than two inches to the right of the point of apex-pulsation.

Fauvel, as already stated (p. 196), was the first observer who fully identified the rhythmical peculiarities of presystolic murmur at the apex. His memoir was published in 1843. Nine-

* *Edinburgh Medical Journal*, November, 1871.

† *Lancet*, March 2nd, 1872.

‡ See p. 214.

teen years earlier, however, Bertin recognized it, and actually founded thereon the positive diagnosis of left auriculo-ventricular contraction.* In one case (*XLIX^e Observation*), he describes the murmur as diastolic in rhythm, accompanying the contraction of the auricles (which, in his estimation, coincided with the second sound), and as attended with *frémissement*. This was, no doubt, an example of the prolonged (third) form of murmur, modified by suppression of its terminal portion as just explained, indicating extreme contraction of the mitral orifice. Its placenary quality may be referred to as confirmatory of this view.

In a second case (*LI^e Observation*), he describes the murmur as "preceding the pulsations of the ventricles;" thus indicating that its rhythm was presystolic. In both these cases the mitral orifice was found, on *post mortem* examination, to be contracted in an extreme degree.

That Hope, fifteen years after the publication of Bertin's book, failed to identify the murmur indicative of mitral obstruction, is sufficiently manifest from his having described it as "attending the ventricular diastole and second sound;" as "always very feeble, soft, like the bellows-sound, and usually on a rather lower key than a whispered *who*;" and as "exceedingly rare." He adds: "I have never known purring tremor accompany a diastolic (*i.e.*, presystolic) mitral murmur, the current being too feeble to produce it."† He would account for the circumstance that Laennec had regarded this murmur as by no means rare, by assuming that he had mistaken for it the murmur of aortic regurgitation. I incline to believe that the preceding reference to his own writings is not unlikely to raise a suspicion in the minds of many, that he has himself fallen into the error which he imputed to Laennec.

The typical presystolic murmur may be simulated more or less closely, as already stated,‡ by

- (a) Pericardial friction-sound of single and systolic rhythm, confined to the area of the apex;
- (b) Prolonged systolic murmur replacing the first sound at the

* *Traité des Maladies du Cœur*, 1824, pp. 176 and 186.

† *Diseases of the Heart*, third edition, 1839, p. 387.

‡ See p. 217.

apex, and extending over the short pause; the second sound being associated with impulse;

(c) Reduplicated first sound audible at the apex.

Since the above was written, I have been enabled to supplement the list of possible sources of error by the addition of,

(d) Postdiastolic murmur of aortic origin transmitted to the apex; and

(e) Prediastolic basic murmur transmitted to the apex; the second sound being accompanied by impulse.

Cases 4 and 6 afford good examples of pseudo-presystolic murmur arising from pericardial friction. The differential diagnosis of the pseudo-murmurs (a), (b), and (c), has been already discussed;* that of (d) and (e) may be now briefly pointed out. The former is well exemplified in the following case. A young man of nervous temperament, and the subject of repeated attacks of rheumatism, was received into hospital on the 21st of August, 1874. The radial pulse was slightly visible, the impulse of the heart strong, and the apex-pulsation felt in the nipple-line. A faint murmur of postdiastolic rhythm was heard at the apex, which corresponded to that described at p. 904, as an abbreviation of the third form of presystolic murmur. At the right base, however, and in the ascending aorta, the second sound was sharp, and was succeeded by a loud bellows-murmur which was traceable to the apex, where it presented the characters already mentioned. The murmur was thus shown to be aortic, not mitral, in origin. Case 89 affords an example of the same kind, but more complicated. Prediastolic basic murmur, audible at the apex by transmission, may be mistaken for presystolic murmur, if, in the latter situation, the second sound be accompanied by impulse; thus assuming the association, and apparently the rhythm, of the first sound. The existence, however, of prediastolic murmur at the base, traceable to the apex, with progressively decreasing intensity, will readily establish the distinction. Case 98 exemplifies these remarks.

Although presystolic murmur, as previously stated, is pathognomonic of mitral stenosis, it is liable to be mistaken for more

* See pp. 217 and 218.

ordinary phenomena, and even when identified, it is often misinterpreted.

Doctor Alexander Silver, of the Charing Cross Hospital, in a thesis of great merit,* remarks that owing to the altered character of the first sound at the apex in mitral constriction, and to its close resemblance to the second sound, which is not here audible, whilst the presystolic murmur covers the normal long pause, the latter is often mistaken for a murmur of systolic rhythm replacing the first sound, the real first sound being mistaken for the second.

I have no doubt this mistake has been, and is still, frequently committed in cases exhibiting the combination of signs mentioned by Dr. Silver; but the misleading concurrence is by no means so common as he has been led to conclude from the observation of the case, so fully and so graphically reported in his monograph. In the greater number of examples of mitral stenosis, the long pause is *not* entirely covered by the presystolic murmur; the second sound is *not* always inaudible at the apex; nor does the first sound resemble the normal second sound in all cases.†

Doctor Silver would regard a sharp first sound, unaccompanied by systolic murmur, as indicative of the "funnel-shaped" form of narrowing; the peculiar character of the sound being, in his opinion, due to the collision of the valve-segments "by surfaces, not by edges." Dr. Whyte Barclay would, on the other hand, interpret a prolonged presystolic murmur in the same sense.‡ I do not think the particular form of narrowing actually present can be affirmed, either from the character or accompaniments of the first sound, or from the length of the murmur. Cases 104 and 111 show conclusively that a sharp and clear first sound at the apex, unaccompanied by murmur, may be associated with mitral narrowing of the button-hole character.

Finally, in reference to differential diagnosis, it is necessary to observe, that when mitral inadequacy coexists with mitral obstruction, the murmurs indicative of the twofold lesion run into

* Prize Essay, *Medical Times and Gazette*, March 16th and 23rd, 1873.

† See Cases 102 to 116, inclusive.

‡ *Loco citat.*

one another, constituting a single prolonged murmur. This may be, and no doubt in many instances has been, regarded as entirely systolic. But the harsh character of the presystolic, contrasted with the usually soft and blowing quality of the systolic element, and the coincidence of impulse and of carotid pulsation with the latter, will render the distinction not difficult.*

Professor Purser thinks it "possible that a powerful auricle, a dilated ventricle, and an orifice (mitral) of normal size, might be as effective in producing a murmur (presystolic) as a ventricle of normal size and a narrowed orifice. In each case the due relation between the size of the orifice and the size of the ventricle being disturbed in the same direction."† But such, in fact, is not the case. No example has been recorded and verified by dissection, of presystolic murmur arising from dilatation of the left ventricle, or occurring in the absence of positive mitral narrowing. Dilatation of the left ventricle, from incompetency of the aortic valves, is of very common occurrence, yet, presystolic murmur is not associated, even exceptionally, with that condition. Theoretically, I would say, that the deranged relationship which determines presystolic murmur, is not that between the mitral orifice and the left ventricle, but that between the volume of blood descending from the left auricle and the force with which it is propelled, upon the one hand, and the diameter of the mitral opening, upon the other. I may add that, as a matter of fact, I have never satisfied myself of the existence of presystolic murmur, where, on *post mortem* examination, I have not found the mitral opening absolutely narrowed.

Amongst the morbid changes directly due to obstruction at the mitral orifice, sanguineous engorgement and extravasation in the lungs is one of the most common. Hope remarks that he had met with pulmonary apoplexy more frequently in connexion with great contraction of the mitral orifice, with or without hypertrophy of the right ventricle, than in any other association.‡ Intercurrent expectoration of pure florid blood is rarely absent in the course of the disease. It is of frequent occurrence

* See Cases 102, 110, and 113.

† *Irish Hospital Gazette*, March 15th, 1873.

‡ *Opus citat.*, p. 249.

in the greater number of examples; and, in the early stages of the disease, as a means of relieving the temporary congestion of the lungs, and as being unlikely, at this period, to be attended with extravasation into the pulmonary structure, it need not be deprecated;* neither should it be the occasion of alarm to the patient. The quantity discharged is usually moderate, and in no instance have I known it to be the cause of sudden death. When hæmoptysis occurs, however, within the few last days of the patient's life, it may be regarded as an infallible indication of pulmonary apoplexy; it is then accompanied by great respiratory distress, general venous engorgement, and dropsy. Hæmoptysis has occurred in 44 out of my 81 cases of mitral obstruction; and of the 15 fatal cases, pulmonary apoplexy was found to have existed in 8.

The term "apoplexy," as applied to these extravasations in the tissue of the lungs, is not only awkward, but inappropriate. That of "hæmorrhagic infarction," first applied by Virchow,† is much preferable. According to him, the condition so designated is one of extreme capillary engorgement and extravasation into the vesicular and interstitial structure of separate lobules of the lung, consequent upon embolism of the branch of supply from the pulmonary artery to these lobules. The vessels beyond the plug being shut off from the pressure of the right ventricle, tension within them is lowered, and there is necessarily an afflux to them from neighbouring capillaries by collateral pressure. The blood so entering the lobule, being no longer under the direct influence of the heart, cannot be expelled. Stasis and capillary hæmorrhage are the result, coagulation extending into the veins.

Lefevre holds that there is simple engorgement, by paralysis, of the capillary walls;‡ whilst Feltz, on the other hand, maintains that infarctus is characterized by actual extravasation.§

Ludwig, as quoted by Niemeyer,|| considers that an explanation may be found in simple stasis, and precipitation of the blood-

* See Case 104.

† Traube's *Beitrage zur Experimentellen Pathologie, u. Physiologie*, Berlin, 1846, and Froiep's *N. Notizen*, Nr. 794.

‡ *Etude Physiologique et Pathologique sur les Infarctus Visceraux*, Paris, 1867.

§ *Traité Clinique et Expérimentale des Embolies Capillaires*, Strasburg, 1870.

|| *A Text-Book of Practical Medicine*, 1869, vol. i., p. 156.

corpuscles within the capillaries, consequent upon the reduction of vascular pressure arising from contraction of the main artery. But, on this hypothesis, the actual occurrence of extravasation by which pulmonary apoplexy is distinguished, cannot be accounted for. The objection to Virchow's theory, that it is based upon an assumption which anatomy does not sanction, namely, that of an anastomosis between the pulmonary plexuses of adjacent lobules, seems fatal to it. It is, however, possible that a collateral circulation, adequate to the production of capillary engorgement and hæmorrhage within the lobule, may be indirectly established through the bronchial system of vessels, which Dr. Waters has shown to be in direct communication with the pulmonary veins.*

This view as to the pathology of sanguineous infarction of the lungs will satisfactorily account for the multiple, circumscribed, and nodular form in which it is presented; whereas, these characteristics could not be explained under any theory implying general and equal vascular pressure in the lungs, whether *a tergo* in the pulmonary artery, from an hypertrophied right ventricle, or *a fronte* in the pulmonary veins, from obstruction or reflux at the mitral or the aortic orifice.

Assuming, as I do, with the qualification above mentioned, that Virchow's doctrine is correct, I should expect to find hæmorrhagic infarction of the lungs most frequently, if not invariably, accompanied by thrombosis of the right side of the heart. Clots have been actually found in the right chambers in 11 out of the 15 foregoing fatal cases of mitral stenosis; and in 8 of the former, pulmonary apoplexy likewise existed. Hence, hæmorrhagic infarction of the lungs was, in every instance, accompanied by thrombosis of the right chambers of the heart. The subjoined case, which has come under my notice whilst the preceding paragraphs were being written, lends considerable support to the doctrine of Virchow. Indeed, it seems to me to establish that doctrine conclusively. The case is given in abstract, as follows:

Agnes F., a child nine years old, was admitted into hospital on the 10th of August, 1874, under my colleague, Dr.

* *The Human Lung*, 1860, p. 201.

Nixon. She was suffering from renal anasarca following scarlatina. A few days after admittance, she suddenly became hemiplegic on the left side. The paralysis was exclusively motor, and only partial in degree. She quickly recovered the use of the affected limbs; but the anasarca continued, despite active treatment. The urine was loaded with albumen, and exhibited epithelial tube-casts. About the middle of August I took charge of the girl, in the absence of Dr. Nixon, and for nearly a month subsequently she continued under my observation. During that time she had an attack of pleuritis with effusion on the right side, from which she made a good recovery. She continued, however, weak and pallid; the feet were slightly swollen, albumen was still present in the urine, and the pupils were persistently dilated. Her condition fluctuated, even from day to day, between extreme debility with partial coma, and a bright and cheerful manner, accompanied with perfect intelligence. Dr. Nixon resumed charge of her about the 6th of September; she was then moribund, and on the 10th she died comatose. The body was carefully examined by Dr. Nixon, to whom I am indebted for the report of the *post mortem* examination, as well as for the favour of obtaining the subjoined engraving. There was considerable effusion into the right pleural cavity, and likewise into the pericardium. The heart was structurally normal, but the right chambers were occupied by a decolorized and friable thrombus. The superior lobe of the left lung exhibited, in its antero-inferior portion, a mass of hæmorrhagic infarction as large as the last joint of the thumb. It was dark, well defined, and perfectly solid; and on section it presented the appearance of black-currant jelly. The two branches of the tertiary division of the pulmonary artery leading to this mass, which included several lobules of the lung, were full, to distension, of decolorized and disintegrated fibrin similar to that found in the right ventricle. The primary division of the pulmonary artery, the tertiary branch leading to the infarcted lobules, and all the adjacent branches of that vessel, as well as those of the pulmonary vein, were empty and collapsed, whilst the lung-structure was elsewhere normal. The condition just described is well represented in the woodcut. (Fig. LIX.)

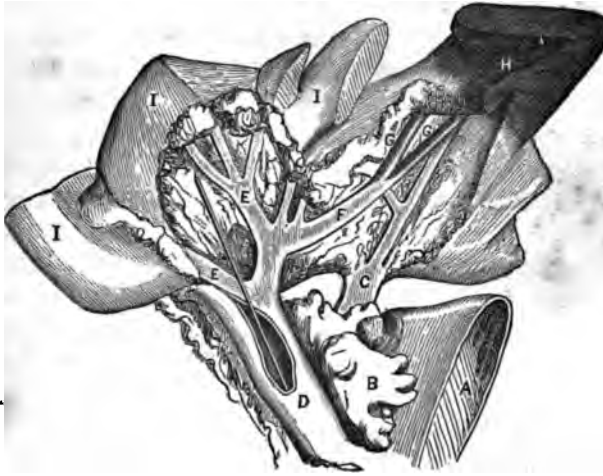


FIG. LIX.

Hæmorrhagic infarction in left lung. (Agnes F.)

- A. Section of left auricle.
- B. Appendix of left auricle, distended by a thrombus.
- C. Left superior pulmonary vein quite empty.
- D. Superior branch of primary division of left pulmonary artery. A bristle is represented as introduced into an opening made in the vessel.
- E. Secondary branches of pulmonary artery empty and collapsed.
- F. Tertiary branch leading to infarcted vessels.
- G. Quaternary and infarcted branches of pulmonary artery.
- H. Infarcted lobules.
- I. Healthy lung-substance.

Dropsy, as previously stated, is of exceptional occurrence in the course of the disease, preceding the final illness; it is then, however, all but invariably amongst the lethal symptoms, and is general. Gangrene of the lower limbs I have not witnessed in more than one instance (Case 114). In this latter respect, disease of the mitral valves, of whatever kind, contrasts strikingly with valvular lesion at the orifice of the aorta. This difference is, no doubt, due to the frequent association of general arterial disease with the latter affection.

With regard to the chambers of the heart, the left auricle is

invariably dilated and hypertrophied, its lining membrane thick and opaque, and the orifices of the pulmonary veins much dilated. I have not witnessed aneurism of the left auricle, of which Thurnam has collected nine examples, in connexion with mitral narrowing.* The left ventricle, on the contrary, is always normal as to capacity and thickness, where mitral inadequacy or either form of aortic valve-lesion is not likewise present, and no general cause of hypertrophy or dilatation exists. Hope was certainly in error in stating that "the left ventricle is sometimes rendered hypertrophied by a contraction of the mitral orifice."†

Doctor Adams, who wrote on this subject three years before the first edition of Hope's book appeared,‡ more correctly stated that in cases of mitral contraction, whilst the left auricle is dilated and thickened, and the pulmonary veins and right chambers are likewise dilated, the left ventricle is rather diminished in capacity.

In no instance which has fallen under my notice has hypertrophy or dilatation of the left ventricle been fairly attributable to mitral obstruction. In the progress of the disease the right ventricle is invariably dilated, and sometimes hypertrophied, whilst the right auricle is dilated and thinned.

The *treatment* of obstruction at the mitral orifice resolves itself into that of the symptoms and sequelæ of the affection. In this respect, however, much may be accomplished by well directed measures, even in the advanced stages of the disease, towards the alleviation of suffering and abatement of urgent symptoms. The mechanical impediment to the circulation once established, is not removable by medicine. The heart is, however, in an eminent degree capable of adapting itself to the altered circumstances under which it is required to function, when, as is usually the case in regard to mitral narrowing, the progress of the disease is slow and regular. The object of treatment at this period should be, therefore, to favour the process of self-adjustment in the heart, by the avoidance of over exertion, and of mental excitement.

If the heart should exhibit evidence of weakness by intermit-

* *Medico-Chirurgical Transactions*, vol. xxi.

† *Diseases of the Heart*, p. 250.

‡ *Dublin Hospital Reports*, vol. iv., 1827.

tent or irregular action, quick and feeble pulse, or by threatened syncope, it should be promptly strengthened by the administration of digitalis, either alone or in combination with iron.

Whilst on this subject, I cannot avoid quoting the pertinent and philosophic remarks of Dr. Foster, of Birmingham. In reference to the compensating power of the heart, he writes: "The propelling power must be augmented, to drive the blood at a greater speed, or the time for its passage must be lengthened.

"When the narrowing is not very great, and the demands on the circulation by exercise are limited, a very perfect compensation is effected by the hypertrophy and dilatation of the left auricle and the right ventricle. By these means the current of blood through the lungs is made more rapid, the tension in the auricle is increased, and the velocity of the current filling the left ventricle is so multiplied, that the balance of the circulation is fairly maintained, in some cases for years. But when the narrowing is considerable, or some unusual strain has disturbed the unstable equilibrium on which the health of these patients depends, then the compensation which the auricle and ventricle offer fails. The rapid irregular pulse tells of the varying quantity on which the ventricle contracts. Sometimes the interval between the ventricular contractions is so great, and the ventricular charge is so small, that the systolic wave does not reach the wrist. Under these circumstances the second indication must be followed; the auricle must have more time to fill the ventricle. This, to say nothing of the increased power given to the cardiac muscle, is exactly what digitalis effects. By slowing the action of the heart, the period of time during which the blood from the distended auricle can flow into the ventricle is increased, and as the extra time allows more blood to pass through the narrowed mitral orifice before the final effort of the auricle is made, that effort is made on a smaller quantity of blood, and is, consequently, more effective; for the smaller the quantity of blood which the auricular muscle has to push before it, the greater will be the velocity given to the current. The ventricle, though contracting less frequently, contracts more effectually. Instead of eighty or ninety irregular contractions per minute, no two succeeding ones equal in force, and some so

valueless that they are not perceptible at the wrist, we get some sixty steady equal beats; the pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognizes its master, and beats a sullen retreat."*

I usually prescribe ℥xv of the tincture of digitalis, with an equal quantity of tincture of the perchloride or acetate of iron, and ℥x of spirit of chloroform, to be taken in an ounce of water every third hour. If pain exist near the apex, or to the inner side of the nipple, I regard it as evidence of debility, tension, and parietal congestion of the right ventricle or left auricle, as the case may be, and proceed on the same principle to exhibit digitalis and iron, or quinine and strychnine, grs. ij of the former with ℥ij of the Pharmacopœial solution of the latter, every third or fourth hour. At the same time, I direct one or two leeches to be applied at the seat of pain, and subsequently an opiate plaster.

Moderate and healthful exercise should be enjoined, in order to promote nutrition, and prevent hepatic and portal congestion. It should not, however, be carried to the point of fatigue, or excitement of the circulation. The food should be selected with special reference to its nutritive and digestible qualities, and no less with a view to the avoidance of flatulence which, by making upward pressure upon the heart and lungs, may give rise to palpitation and dyspnoea. The bowels should be kept free by means of aloetic and saline aperients; and alcohol, except in very small quantity, and by preference in the form of good claret, should be forbidden. Malt liquors are especially liable to disagree with the patient, owing to their tendency to cause portal congestion. Copious imbibition of liquids is to be prohibited, because the volume of the blood is thereby, for the time, increased. For this reason, condiments which provoke thirst must be avoided.

Hæmoptysis, in moderate quantity, need not cause alarm. The condition which it indicates, namely, congestion of the lungs,

* *British and Foreign Medico-Chirurgical Review*, No. xcv., July, 1871; and *Clinical Medicine*, 1874, p. 101.

and engorgement of the right side of the heart, is best treated by active purgation, dry-cupping the chest, and, if the discharge be copious or persistent, by the use of ergot or ergotin. ℞xx-xxx of the liquid extract of ergot may be given in half an ounce of cold water every second hour, till the hæmorrhage cease, or gr. ss of ergotin in solution may be injected hypodermically at corresponding intervals, till the object is attained. The latter is decidedly the more efficacious method of employing this agent.

The treatment of dropsy, so common in the advanced stages of the disease, should be directed, in the first instance, to the relief of congestion of the kidneys, which are functionally impaired by sanguineous engorgement. Six to eight ounces of blood may be taken from the loins by cupping or leeching; or, if the patient be very weak, dry-cupping alone should be practised. In either case a succession of poultices to the loins, as warm as can be borne, should be maintained. The bowels should, at the same time, be well moved by means of compound powder of jalap, grs. xxx, with grs. ij of calomel, in the form of electuary; or, ʒij of the acid tartrate of potash dissolved in a wineglassful of the infusion of senna, with ʒj of the tincture. It may be necessary to give sedatives in order to induce sleep; for this purpose, grs. xx of chloral hydrate, or grs. xv of this, with ℞x of liquor of the hydrochlorate of morphia, may be given in an ounce of water at bed-time.

If the nature of the case be early recognized, and the patient judiciously and continuously treated as here recommended, a protracted period of moderately good health, and even of social enjoyment, may be confidently guaranteed. The patient should, however, be warned that indiscretion of any kind, especially such as may induce acute congestion of the lungs, or otherwise overtax the power of the heart, is likely to be followed by serious consequences.

Amongst the succeeding cases are many which, thus treated, have continued for several years, little if at all, aggravated by lapse of time.

In fine, it may be averred that this is, *par excellence*, the form of organic disease of the heart which is most amenable to early

and appropriate treatment, so far as refers to grave consequences and premature death.

The following 16 cases will be found to illustrate the preceding remarks, and to present, in epitome, the history and pathology of mitral stenosis. They have been all under observation and treatment for some time, most of them for a protracted period; and, finally, a careful *post mortem* examination of the body was made in every instance.

The cases which were not fatal, 66 in number, are presented in summary in Tables XI. and XII., and are explained, where necessary, by marginal notes. The fatal cases are likewise included in these Tables, with a view to obtaining complete results under the several heads mentioned.

CASE CII.—*No History of Rheumatism; Orthopnoea and Palpitation; Œdema; Quick and Faltering Pulse; Strong Impulse; Hæmoptysis; Pain at Precordium and Extending down Left Arm; Angina; Rough First Sound with Presystolic Murmur at Apex; Soft Systolic Murmur at Base; Reduplication of Second Sound; Subsequent Development of Systolic Murmur at Apex; Suppression of Presystolic Murmur for One Day, and of Radial Pulse during the Last Week of Life, with very Slow Respiration; Coma; Death. Autopsy: Effusion into Pericardium; Dilatation and Thrombosis of Right Chambers of Heart, and Dilatation of Tricuspid Orifice; Dilatation and Hypertrophy of Left Auricle; Great Contraction and Funnel-shaped Transformation of Mitral Orifice; Emphysema and Sanguineous Infarction of the Lungs.*

Anne K., aged twenty-eight years, a seamstress, was admitted into hospital on the 24th of November, 1865. Never had rheumatism. Has repeatedly spat blood. Three years previously, she complained, for the first time, of palpitation and breathlessness after unwonted exertion of any kind. Two years later, she had cough; and for four months preceding admittance, she had been unable to lie down, owing to the difficulty of breathing which the attempt to do so occasioned. Her feet had been swollen for the last month.

The face was somewhat congested, and the eyelids puffed, but there was no lividity of the lips or extremities. Pulse rapid, extremely weak and faltering; lower extremities œdematous. The cervical veins were enlarged, and distended, and the external jugular pulsated synchronously with ventricular systole, but the lightest pressure above the clavicle promptly arrested this pulsation. The chest was abnormally resonant, and permanently distended, as in the state of inspiration; the impulse of the heart was distinct and vigorous.

The apex pulsated at a point rather internal to the usual position. At this point the first sound was rough; it was immediately preceded by a harsh murmur which extended up to, but did not implicate it, and was loudest at the apex. At midsternum, a soft, localized, and accompanying systolic murmur existed. The second sound was clear, but reduplicated at midsternum.

The following diagnosis was made: Mitral stenosis, hypertrophy of left auricle, dilatation of both right chambers and of right auriculo-ventricular opening, pulmonary emphysema.

On the 17th of December there was hæmoptysis, the blood discharged being florid, and mixed with frothy mucus; and on the 31st the patient complained of acute pain located in the region of the heart, and extending down the left arm. This pain was anginal in character, being accompanied by a sense of constriction in the chest, and by great respiratory distress.

About the middle of January, a well pronounced systolic murmur was developed at the apex. It was continuous with the presystolic murmur, constituting an appendix to it, but distinguishable from it by its less harsh quality, and by its rhythm. On the following day the presystolic murmur was suppressed; it was not again heard, whilst that of systolic rhythm continued audible up to the patient's death.

The radial pulse was entirely suppressed during the last week of the patient's life; still she continued to move about, and declared she felt better. Two days before her death, which took place on the 26th of January, she expectorated a large quantity of dark blood; respiration, at the same time, fell to 15 in the minute, at which rate it continued to the end. She died comatose.

The *post mortem* examination revealed the following condition of organs: Both lungs were emphysematous, and in the apex of each there was an insulated and well defined mass of very dark effused blood. These portions of the lungs were indurated and non-crepitant. The bases of the lungs were slate-coloured, and contained some extravasated blood; and on the surface of both there were several emphysematous projections filled with solidified blood. The pericardium contained six ounces of serum.

The heart, especially on the right side, was greatly distended with dark clotted blood; it had lost its natural figure, and become greatly expanded transversely. The right ventricle was considerably dilated, and its walls were slightly thickened. The right auriculo-ventricular opening was very large; but its valves, though incompetent, were healthy, as were likewise those of the pulmonary artery. The left auricle was large, its walls were much thickened, and the orifices of the pulmonary veins were slightly dilated. The left ventricle was rather below the normal size; its walls were not thickened. The mitral orifice was much reduced in diameter. Both segments of the mitral valve were thickened and united in such a manner as to form a funnel-shaped passage about an inch and a-quarter long, and projecting into the left ventricle; the opening at the ventricular end of this passage barely admitted the point of the little finger.

A loose and pendulous flake of fibrin was attached to the auricular surface of one of the valve-segments, in such a position that, during life, it must have vibrated in the influx-current from auricle to ventricle.* The aorta and its valves were perfectly sound.

* See *Proceedings of the Pathological Society of Dublin*, vol. ii., new series, 1865, p. 179. I do not now hold the opinion then expressed, that the vibration of this shred of fibrin was the cause of the presystolic murmur mentioned in the report of the case.

CASE CIII.—*Rheumatism ; Intemperance ; Mechanical Injury in Region of Liver ; Hæmorrhoids ; Dyspnœa and Lividity ; Rapid and Feeble Pulse ; Anasarca ; Ascites, and Pleural Effusion ; Legs Punctured ; Diminished Secretion of Urine ; Hæmoptysis ; Gangrene of Feet ; Death. Autopsy : Ascites ; Liver Contracted, and in "Nutmeg" Condition ; Spleen Thickened in its Capsule, and containing Masses of Mortar-like Substance ; Bases of Lungs Congested ; Effusion into Pleura and Pericardium ; Thrombosis of Right Auricle ; only two Valve-Segments at Right Auriculo-Ventricular Orifice ; Hypertrophy of Left Auricle ; Great Contraction, and Button-hole Figure of Mitral Opening ; Aortic Valves Slightly Inadequate.*

Jane G., aged thirty-four years, a native of Liverpool, and of intemperate habits, was received into hospital on the 26th of January, 1866. Whilst crossing over to Dublin, six weeks previously, she was exposed to wet and cold on the deck of a steam-boat, and on her arrival her breathing was much embarrassed, and her feet and legs became swollen. The poor woman informed me that at the age of thirteen she had an attack of rheumatic fever, from which, however, she suffered no permanent impairment of health ; she occasionally suffered from bleeding hæmorrhoids. Three years prior to admittance, she received from her husband, whilst he was drunk, a blow with the clenched hand in the region of the liver. She was knocked down by the blow, and suffered great pain in the abdomen for some time afterwards. Somewhat later she had ascites and anasarca, and for the latter her legs were punctured. After recovery from this attack she felt her respiration permanently embarrassed.

State on admittance : Face, especially lips, quite livid, and conjunctivæ injected with dark blood ; extremities cold and livid, and feet and legs highly cedematous and tense ; several minute openings had formed spontaneously on the legs, most probably in the seat of former punctures, from which serum freely exuded. Pulse rapid and very feeble, but regular. No visible pulsation of arteries to be anywhere detected. Urine reduced to three ounces in twenty-four hours, and coloured with

bile pigment. There was partial effusion into both pleural cavities.

The impulse of the heart was strong, and attended with *frémissement*. The apex pulsated in the normal position, and at this point two murmurs were audible; viz., a loud and harsh presystolic murmur, which was immediately succeeded by a clear first sound, accompanied by a soft and blowing diastolic murmur. The former was confined to the area of the apex, whilst the latter was derived from the base, where it was loudest, and manifestly of aortic origin; it was traceable upwards in the course of the aorta for a distance of about two inches.

The liver projected considerably below the costal cartilages; it was firm and even on the surface. There was considerable effusion of serum into the peritoneum. The patient's fingers were distorted, and the joints enlarged, apparently from chronic gout.

The legs were again punctured, but they yielded very little serum; and on the following day patches of incipient gangrene appeared upon the dorsum of either foot. The patient was now pulseless. In the course of the succeeding night she coughed a good deal of dark blood, and died at 1.30 A.M.

The body was examined thirteen hours after death, with the following result: The liver was contracted, and in the "nutmeg" condition; its investments, both serous and fibrous, were much thickened. The spleen was of normal size; its capsule was opaque and greatly thickened, and in its parenchyma two large masses of a yellow putty-like substance were found, which seemed to consist of fat and urate of soda; kidneys healthy. There was a considerable effusion of serum into both pleural cavities. The bases of both lungs were solid, and the investing pleuræ were dense and opaque. The pericardium contained a pint of straw-coloured serum, in which a large shred of false membrane floated loosely, having no attachment to either surface. The heart appeared as if daubed over with white paint; it was, however, smooth and glistening on the surface, and of average size.

The right auricle contained a large thrombus, which extended to the auriculo-ventricular opening, through which it projected

by a round bulbous extremity; it did not, however, extend further. The right ventricle was scarcely altered, but the auriculo-ventricular valve was *bicuspid*. The left auricle was much thickened and dilated. The left ventricle was rather reduced in capacity, but thickened. The mitral orifice was of a button-hole figure, and greatly reduced in size, scarcely admitting the tip of the little finger. The mitral valve was thick, rigid, and all but completely calcified; it projected into the ventricle, but the segments lay evenly together so as to close the opening. The aortic valves were thick and rigid; they admitted of slow leakage into the ventricle when water was poured into the aorta above them. The lining membrane of the ascending portion of the arch of the aorta was red; it was likewise dotted with yellow patches of atheroma in an early stage of formation.

Measurements: Right auricle normal; right ventricle, walls $\frac{5}{8}$ of an inch thick at apex, and $\frac{3}{8}$ at middle; left auricle, walls $\frac{1}{4}$ of an inch thick in sinus; cavity, measured from septum to outer wall, $2\frac{1}{4}$ inches, from superior wall to mitral valve, $2\frac{3}{4}$ inches, from anterior to posterior wall, 3 inches; left ventricle, walls $\frac{5}{8}$ of an inch thick at apex, $\frac{1}{2}$ an inch at middle, and $\frac{3}{8}$ of an inch at base; cavity, measured from root of mitral valve to apex, $2\frac{1}{2}$ inches, from anterior to posterior wall, $1\frac{1}{2}$ inch; ascending aorta, 1 inch in diameter.*

CASE, CIV.—*History of Scarlatina; Cough, and Repeated Hæmoptysis; Dyspnœa; Weak and Failing Pulse; Anasarca and Ascites; Apex-point Displaced Outwards; Presystolic and Systolic Murmur; Sounds of Heart Sharp and Clear; Sudden Death. Autopsy: General and Firm Adhesion of Pericardium; Slight Dilatation and Thickening of Right Ventricle; Dilatation of Tricuspid Opening; Great Dilatation and Hypertrophy of Left Auricle; Extreme Contraction of Mitral Orifice; Left Ventricle Normal; Substance of Heart Healthy; Aorta of Small Calibre, and Pink Internally; Valves all Healthy, except Mitral.*

Thomas N., aged ten years, admitted February 3rd, 1868.

* See *Proceedings of the Pathological Society of Dublin*, new series, vol. iii., part i., p. 22.

Five years previously, he had scarlatina, and spat blood. When admitted, he had cough with mucous expectoration; respiration was quick, and the pulse was scarcely perceptible. He spat blood about once every three weeks, the hæmoptysis continuing three days, and being always attended with considerable alleviation of his suffering. The most urgent symptoms were weakness, and shooting pain in the region of the heart; and the chief physical sign was presystolic murmur at the apex, which pulsated immediately inside the nipple-line. He remained under treatment till the 9th, when he was discharged at his own request, much relieved, but without any change of the apex-murmur. The patient presented himself at the hospital on the 17th of February, and again, on the 23rd of March.

On the latter occasion the following note was made: Radial pulse not perceptible; cardiac pulsations 120 in the minute; respirations 36. There has been no hæmoptysis for the last few days; but it may be brought on at once by exertion, such as fiddling with carpenter's tools, to which he is much addicted. Cough continues through the night, and almost entirely prevents sleep. Appetite failing, and stomach irritable; chest prominent, and supra-clavicular fossæ obliterated. Both a presystolic and a systolic murmur are now audible at the apex, which pulsates in the nipple-line. To have a sinapism to chest, and an anodyne expectorant. He continued to spit blood in large quantity, and was re-admitted on the 22nd of June. He was then in a state of unconsciousness; his face puffed and pallid, lips livid, and no pulse perceptible at the wrists.

He was universally anasarcaous; the abdomen was distended with liquid, and there was strong and heaving pulsation of the carotids on both sides. Respiration was accompanied with coarse crepitus. Precordial dulness was extended, and the impulse of the heart was prolonged and heaving. At the apex, a faint but rough presystolic murmur was heard; it was followed by a sharp clicking first sound. The second sound was heard at the apex after every second or third pulsation only; it was clear, but less sharp than the first sound. To have, thrice daily, a draught containing ℞ of chloric ether, spirit of nitrous ether, and aromatic spirit of ammonia respectively; wine, and light nourishment.

25th. He died suddenly at 1 o'clock to-day. The body was examined the following morning. The pericardium was universally and firmly united to the heart by old adhesion. The right ventricle was thickened and dilated, and the tricuspid orifice enlarged. The left auricle, especially the appendix, was greatly dilated and thickened. The left ventricle was small, and normal as to thickness of walls; its substance was firm.

The mitral orifice was reduced to the size of the barrel of a goose-quill; the valves were adherent, except in the centre, where an oval opening of the above mentioned size existed; the edges of this opening were seamed and regular.

The aorta was small, and its lining membrane was of a pink colour which was not removable by washing; the aortic and pulmonary valves were healthy and competent.

CASE CV.—*Fright after Child-birth, followed by Jaundice; Hæmoptysis; Presystolic Fremitus and Murmur; Reduplication of both Sounds of Heart; Intercurrent Pericarditis; Typhus Fever; Death. Autopsy: Hydro-pericarditis, and Roughening of Surface of Heart by Recently Effused Lymph; Enlargement of Heart; Dilatation of Tricuspid Opening; Dilatation and Thickening of Left Auricle; Great Narrowing of Mitral Orifice, and Thickening of Valves and of Tendinous Chords; Liver Olive-Coloured and Fatty; Biliary Ducts Dilated, but not Obstructed; Scirrhus of Pancreas.*

Maria F., aged thirty years, admitted June 18th, 1868; has had one child, which is five months' old. One month after confinement she was frightened by the abrupt entrance of a strange man into her room whilst she was in bed. Four days after this occurrence her friends noticed that she was jaundiced. The jaundice was not attended with pain, nor was it preceded by illness of any kind. She is now deeply jaundiced, of a dark olive colour; urine deeply tinged with bile pigment, and not abundant. Nothing noticeable in region of liver, except some slight tenderness in scrobiculus cordis. At the apex of the heart a soft systolic murmur is audible; at the left base both sounds are double, but no murmur is to be heard here.

June 19th. Urine loaded with bile, but less deeply coloured than formerly. Harley's test for bile acids gave a dark opaque stratum, not purple. Has been taking blue pill, taraxacum, and dried soda; also nitro-muriatic acid in infusion of chiretta.

July 1st. Pulse 96, of good volume, and regular. For the last week there has been less of the colouring matter of bile in the urine; complains of a "fluttering" at pit of stomach. On making a careful examination of the heart to-day, I discovered a very distinct and characteristic presystolic murmur at the apex. It is somewhat rough, and faintly audible for a short distance to the right; both sounds are double, but not very distinctly so at the base; impulse of heart in normal situation.

11th. Admits she spat a little blood, but only once, about three weeks prior to admittance. She experiences a sensation in the skin as if "mill-dust were sprinkled over her"; last night she had a sharp stinging pain in the region of the heart.

On July 27th the patient was attacked with acute pain in the head, accompanied with general febrile symptoms. There was tumultuous action of the heart, and a single, loud, grating sound at the apex, which masked the previous murmur. She was at once put under mercurial treatment; a leech was applied to the precordium, and followed by a succession of warm poultices.

August 3rd. Her condition was as follows: viz., Pulse 108, with occasional intermissions for the first time. No increase in extent of precordial dulness. At apex, a distinct *frottement* was perceptible by the hand, and here a loud jarring and superficial murmur was heard, synchronous with the second sound, but likewise covering the greater part of the long pause; it masked effectually the endocardial murmur previously heard. At a short distance to the right a presystolic murmur was faintly heard, manifestly by transmission. No friction or other abnormal sound heard in this situation, both cardiac sounds being here normal. The grating sound was transmitted into axilla from apex, but with progressively decreasing distinctness.

I left town on the evening of August the 3rd, and did not again see the patient till the 11th. She had, in the *interim*, contracted typhus fever of a most virulent character. The maculæ were large and dark, and interspersed with petechiæ. When I

visited her she was comatose; pulse rapid and feeble. She died in the afternoon, and on the following day (the 12th) the body was examined.

The pericardium contained nearly a pint of serum. The heart weighed eleven and a-half ounces. The inferior surface of the right ventricle was somewhat rough and granulated. The right chambers were normal, but the tricuspid orifice was greatly dilated, admitting four fingers introduced edgewise. The left auricle was dilated and thickened. The mitral orifice was surrounded by a dense white cartilaginous rim, formed by the altered valves; it was circular in figure, and admitted only the tip of the little finger. The left tendinous chords were thickened, shortened, and very rigid. The left ventricle was normal. The liver was of the ordinary size, of a deep olive colour, soft, and lardy, and easily broken down with the finger; the gall bladder was contracted and empty. The hepatic, cystic, and common biliary ducts were greatly dilated; the latter readily admitted the end of the thumb, but the entrance into the duodenum was not obstructed. The head of the pancreas was enlarged and hardened; it was dense and rigid on section.*

CASE CVI.—Mechanical Injury of the Chest; Subsequent and Repeated Attacks of Dyspnoea, Palpitation and Collapse; Intercurrent Congestion of the Lungs, and Hæmoptysis; Presystolic, and Subsequently Systolic Murmur at the Apex of the Heart; Soft Systolic Murmur at the Xiphoid Cartilage; Variola; Pericarditis; Thrombosis of the Heart and Sudden Death. Autopsy: Congestion of the Lungs; Partial Pericarditis; Enlargement of the Heart by Hypertrophy and Dilatation of the Right Ventricle; Thrombosis of the Right Chambers; Hypertrophy and Dilatation of the Left Auricle; Great Contraction of the Mitral Orifice; Left Ventricle Normal.

Robert McB., aged fourteen years, and healthy-looking, was received into hospital on the 3rd of September, 1867. He had

* For this and the preceding case, see *Proceedings of the Pathological Society of Dublin*, new series, vol. iv., part i., p. 18.

enjoyed good health; had not had rheumatism, nor had he been otherwise seriously ill up to a year previous to date of admittance, when he was thrown from a pony in the street, and the animal trod upon his chest. This accident was followed by great pain in the chest; also by dyspnoea and expectoration of florid blood.

After a short respite, he resumed his duties as light porter in a grocer's shop, and for the succeeding eight months enjoyed comparatively good health. He was then suddenly attacked with dyspnoea and palpitation of so severe a character that he was obliged to sit down in the street. Seizures of a similar kind occurred from time to time; and, in one of unusual severity, which occurred on the day of admittance, he fell down in the street, and was carried to the hospital in a state of collapse.

When admitted, he was cold and pallid, the lips and extremities were livid, and respiration was rapid and oppressed; the heart pulsated tumultuously; the radial pulse was 150, and very weak.

On the following morning (4th) I examined him for the first time. His condition was then as follows: Pulse 84, and quite regular. Respiration 24. He was still pale, but quite conscious; the lips were livid; the chest was resonant throughout, and respiration was everywhere normal. The external jugular veins pulsated synchronously with ventricular systole. The impulse of the heart was strong; the apex pulsated in the normal position, and with ordinary force. At the apex-point a presystolic murmur existed, of so typical a character as to warrant, on the instant, the diagnosis of mitral narrowing. Both sounds of the heart were distinctly pronounced, and quite clear. At the base of the xiphoid cartilage a loud bellows-murmur of systolic rhythm was heard; it was traceable for two inches upwards in the course of the aorta. Beneath the spine of the left scapula the presystolic murmur, and also both sounds of the heart, were heard. The second sound was slightly intensified in the pulmonary artery. His condition improved so much that he was discharged at the end of a month, free from all urgent symptoms. The treatment consisted in the application of two leeches to the pre-

cordium, and the administration of infusion of the American wild cherry, ʒviiss; with ʒij of chloric ether and of aromatic spirit of ammonia, respectively; a tablespoonful to be taken thrice daily. In the event of a paroxysm of dyspnœa occurring, a draught was to be given, consisting of ʒss of compound spirit of ether, and ℥x of chlorodyne, in ʒj of camphor water.

He had no return of paroxysmal dyspnœa after the first evening of his sojourn in hospital; but he spat blood repeatedly. On one occasion his pulse fell to 60. Before his discharge, the murmur at the ensiform cartilage had ceased to be audible.

He was again admitted on the 27th of November, under Dr. Hughes. In the interim, he had repeatedly spat blood.

He next presented himself on the 1st of May, 1868. Hæmoptysis then occurred whenever he walked quickly; he could lie only on the right side; the second sound was double at base, otherwise the physical signs were as last noted.

During the remainder of the year 1868 he was re-admitted several times for acute congestion of the lungs with bronchitis. In these attacks he suffered from great dyspnœa, and spat blood repeatedly; but when relieved, as he usually was, by a short course of treatment, including tincture of the perchloride of iron with digitalis, he was able to resume his work as a porter.

The intervals between his admissions varied from weeks to months, the longest having been from May, 1868, to August, 1869. During these intervals he occasionally reported himself at the hospital, but enjoyed moderately good health, and was able to perform his duties, which were sometimes laborious.

When he came under my care in August, 1869, the apex of the heart was displaced to the left, and occupied a position in the nipple-line. A presystolic murmur, more harsh than previously, existed at the apex. A systolic murmur was likewise audible in the same situation. After a short course of treatment, his health improved, and he left hospital to resume his work.

He was subsequently admitted on the 2nd of October, 1869, on the 29th of June, 1870, and on the 12th of May, 1871. On these several occasions he was suffering from acute pulmonary congestion, with hæmoptysis, from which, as previously, he ob-

tained prompt and complete relief under rest and ordinary treatment.

He was admitted for the last time in the early part of November, 1871. His most urgent symptoms then were cough and dyspnoea. Whilst in hospital he had two attacks of angina pectoris, which were of the most aggravated character and nearly fatal; he was then suffering from dyspnoea, which was associated with congestion of the lungs, and bronchitis. The apex of the heart pulsated rather feebly half an inch outside the nipple-line, and in the sixth intercostal space. The presystolic murmur was now masked by a loud systolic murmur, which was diffused over the entire front of the chest, but was not transmitted into the carotids.

His general health was much improved, and he was about to be discharged on the 4th of December, when, unfortunately, he contracted smallpox, some cases of which were then in the hospital. For two days he progressed favourably, having been successfully vaccinated in childhood, and his arm presenting two well-formed scars. The eruption was discrete, and attended with very slight fever; it was out on the third day, and on the fifth day the pustules were full. In short, the case, as one of variola, was in every respect of the mildest character. On the evening of the 9th (fifth of the disease), he was suddenly attacked with dyspnoea and faintness; and, when I visited him on the following morning, he was very weak and breathed with great difficulty. The face was congested, the cervical veins turgid, respiration of a gasping character, and the pulse small, but regular; the cardiac phenomena were as last noted.

Examination of the chest afforded no satisfactory explanation of the unfavourable change which had taken place; the lungs were slightly congested at the base, but respiration was everywhere audible, and expectoration was free, and of the character usual in chronic catarrh.

He continued in this state till the 15th, propped up in bed, moaning and gasping for breath. On the morning of that day he died suddenly, and without a struggle, after getting into bed from the night chair.

The body was examined twelve hours after death. The lungs

were voluminous and congested, especially at the base, but no extravasation had anywhere occurred ; both apices were attached to the chest-wall by old adhesions ; a few cicatrices existed in the left apex, and the internal surface of that lung was firmly attached to the pericardium. The sac of the pericardium contained no liquid, but its lining membrane was rough on the right side from recent effusion of lymph, and the corresponding portion of the surface of the right auricle exhibited a similar condition. I believe this was the result of pericarditis developed in the course of the variola. The heart was large, weighing eighteen ounces and three-quarters ; the right ventricle was hypertrophied and dilated ; it formed the greater portion of the apex of the heart. Both the right chambers were quite full of decolorized fibrin, which adhered firmly to the internal surface of the auricle, thence passing through the tricuspid orifice into the ventricle. This was, no doubt, the immediate cause of death. The thrombus had been forming for some days previously, most probably from the 9th, when the unfavourable change above noticed took place, and afforded the only rational explanation of the subsequent symptoms and the sudden death.

The left auricle was dilated and thickened, and the orifices of the pulmonary veins were much enlarged. The left auriculo-ventricular opening was reduced to the size of the point of the index finger ; it was funnel-shaped, opening into the ventricle by a slit-like aperture, and formed by the thickened segments of the mitral valve. The anterior segment was of cartilaginous consistence at the base, and the passage was smooth throughout ; the papillary muscles and tendinous chords were not hypertrophied.

The aortic and pulmonic valves were healthy, and the left ventricle was strictly normal as to capacity and thickness.

The systolic bellows-murmur heard at the ensiform cartilage on the 4th of September, 1867, and repeatedly afterwards, but coinciding on every occasion with systolic pulsation of the jugular veins, was most probably due to tricuspid reflux. This is a phenomenon of very rare occurrence from simple engorgement of the right ventricle. The murmur of the same rhythm and quality which was subsequently developed at the apex, must

have resulted from mitral regurgitation. The displacement of the apex of the heart to the left was due to hypertrophy with dilatation of the right ventricle.*

CASE CVII.—*History of Acute Rheumatism; Cough and Hæmoptysis; Orthopnoea and Œdema; Paroxysmal Dyspnoea; Irregularity of Pulse; Albuminuria; Epigastric Tenderness, and Extension of Hepatic Dulness; Pulmonary Œdema; Displacement of Apex of Heart Outwards; Presystolic Fremitus; Presystolic and Systolic Murmur at Apex; Presystolic Murmur likewise Audible at Left Margin of Sternum; Systolic Murmur at Xiphoid Cartilage; Death by Slow Asphyxia. Autopsy: Serous Effusion into the Pericardium; Enlargement of Heart; Thrombosis of Right Chambers; Dilatation and Hypertrophy of Right Auricle; Slight Hypertrophy of Right Ventricle; Thickening of Tricuspid Valve, and Constriction of Orifice; Dilatation and Hypertrophy of Left Auricle; Great Constriction of Mitral Orifice; Thickening of Papillary Muscles and Tendinous Chords; Left Ventricle Thinned at Apex, but otherwise Normal; Aorta Reduced in Size, Hyperæmic Internally, its Valves Thick and Slightly Incompetent.*

Patrick McD., aged twenty-three years, a shoemaker, was admitted into the Mater Misericordiæ Hospital on the 22nd of July, 1870. He had had rheumatic fever twice; viz., at the ages of nine and thirteen years respectively, and had been partially invalided for the last nine months. Five weeks anterior to the date of admittance he began to cough; he then spat blood for a week; three weeks later his feet began to swell, and for the last week dyspnoea had been so urgent that he could not lie down.

When admitted he was pale, but with a malar blush. He suffered from aggravated dyspnoea, which was occasionally paroxysmal. Respiration was 48. Pulse arhythmically irregular, varying from 96 to 108, and very weak; feet and legs greatly swollen; tongue clean and moist, but livid. Urine passed in small quan-

* See *Proceedings of Pathological Society*, new series, vol. v., part i., p. 11.

tity, 1·020 in sp. gr., and containing a good deal of albumen. The liver was enlarged in both lobes, descending about two inches below the umbilicus, and tender to pressure. There was cedema of both lungs,

The apex of the heart pulsated in the sixth intercostal space, two and a-half inches outside the nipple-line. Here, a presystolic fremitus was perceptible; a coarse and jarring presystolic murmur was audible in the same situation, whilst the first sound was faint, and associated with a soft but feeble murmur.

At the xiphoid cartilage, a soft systolic murmur, somewhat metallic in quality, was heard; it was faintly propagated in the course of the aorta, but was not audible in the neck. The second sound was sharp at the base; it was not double, nor was it accentuated in the pulmonary artery. Tincture of *nux vomica* and quinine were prescribed, and the chest was dry-cupped. No improvement, however, took place, and the patient died on the 27th.

On examination of the body, half a pint of serum was found in the pericardium, together with a few flakes of unattached fibrin. The heart was enlarged, globular in figure, and weighed seventeen and a-half ounces; the visceral pericardium was thickened, opaque, and rough on the posterior surface of the right auricle, and to a slight extent also on the anterior surface of the right ventricle. Two long and pendulous flakes of fibrin adhered to the left apex. The root of the pulmonary artery was highly vascular and opaque on the external surface.

The right chambers contained a good deal of decolorized fibrin. It extended from the auricular appendix, which was filled with it, through the tricuspid orifice, and into the right ventricle; it did not, however, enter the pulmonary artery.

The tricuspid orifice was reduced to the size of the tip of the middle finger; it was ovoid in figure, and bounded by the coherent valve-segments. These latter were greatly thickened, but smooth and white, as were likewise the tendinous chords; the papillary muscles were thickened.

The right auricle was dilated and somewhat hypertrophied; the right ventricle was normal as to size, but thickened; the pulmonary artery and its valves were normal. The left auricle

was greatly dilated, and the segments of the mitral valve were united and thickened, and the mitral opening was converted into a slit which admitted only the point of the index finger; it presented some calcareous patches on the auricular aspect. The left papillary muscles and tendinous chords were likewise hypertrophied, but smooth. The left ventricle was altered only in a very slight degree; it was dilated and thinned at the apex.

The aorta was remarkably small in calibre; it was crimsoned internally; the aortic valves were slightly thickened and incompetent, but even and smooth on the surface.

CASE CVIII.—*History of Rheumatism; Sudden Right Hemiplegia and Aphasia; Rapid, Weak, and Feeble Pulse; No Cardiac Murmur; Death by Exhaustion. Extensive Softening of the Left Anterior Lobe of the Cerebrum; No Embolus Detected; Mitral Orifice Greatly Contracted by Cohesion of the Valve-Segments; Left Auricle Dilated and Thickened; Left Ventricle Normal.*

Jane Q., aged forty-seven years, was admitted into hospital on the 17th of March, 1866. She had had a few attacks of rheumatism, but otherwise she had been healthy up to the 28th December, 1865.

On the night of the 27th December she retired to rest in her usual state of health, and on the following morning it was found that she had lost the use of the right arm and leg, and of the corresponding side of the face; she had also lost the power of speech.

At the date of her admittance, there was complete motor paralysis of the right arm, and partial paralysis of the right leg; she dragged the leg in walking, and she was barely able to draw it up and extend it when in bed. The sphincters were not paralysed, nor was sensibility in either limb affected. The features were drawn towards the left side, and the tongue was protruded to the right.

The patient was, however, capable of closing both eyes, of corrugating the forehead, and of moving the tongue to the left side when directed to do so. She could swallow, and was

quite intelligent, but unable to utter a word beyond "yes" and "no." She moaned incessantly. The pupils were normal and equal in size. The pulse at the wrist could not be registered. The heart pulsated at the rate of 160, very feebly and irregularly. Both cardiac sounds were morbidly clear, and extensively transmitted over the chest; there was no cardiac murmur.

On the evening of the 20th of March, the right side of the face became flushed, its temperature rose, and it throbbed perceptibly, whilst the left side remained pale and cold.

She died on the 24th, having suffered much during the preceding few days.

The body was examined twenty-four hours after death. The brain was of normal consistence and apparently healthy, with the following exception: The anterior lobe of the left hemisphere was much reduced in volume, of a light-yellow colour, and semi-diffuent; its convolutions and sulci were effaced, and the left olfactory convolution and bulb were softened and enlarged, the former having become cylindrical, and been brought to the surface of the brain by obliteration of the sulcus. The brain exhibited no trace of hæmorrhage or of inflammation, and no plug was found in any of the cerebral vessels. The second and third left frontal convolutions were disorganized, as were likewise the left orbital convolutions.

The left auricle of the heart was enlarged and thickened; the mitral orifice was greatly contracted by cohesion of the valve-segments; it admitted only the tip of the index finger. The left ventricle was normal.

No cardiac murmur whatever was detected in this case. The patient was seven days under observation, but during the whole of that period her weakness was extreme.

CASE CIX.—*Palpitation and Cardialgia; Œdema; Hæmoptysis; Feeble and Irregular Pulse; Presystolic and Systolic Murmur at Apex of Heart, the former Suspended for Eighteen Days; General Serous Effusion; Death. Autopsy: Enlargement of Heart, and Fatty Growth on its Surface; Dilatation of Right Auriculo-Ventricular Orifice; Thrombosis of Right Chambers; Great Contraction of Mitral Opening; Dilatation and Hypertrophy of Left Auricle; Left Ventricle Normal; Aortic Valves Healthy and Competent; Extensive Sanguineous Infarction of the Lungs; Disfigurement of the Liver by Early Tight Lacing.*

Jane McG., aged fifty years, was received into hospital, June 1st, 1866. She had not had rheumatism. Two years previously, after suffering much anxiety, she experienced, for the first time, palpitation and pain in the region of the heart; her feet and legs had recently become swollen.

At the date of admittance, her condition was the following: Radial pulse not perceptible; slight œdema at outer ankles; extremities cold and livid; liver much enlarged and prominent, but even on the surface; it descended nearly to the umbilicus; she had not had jaundice; respiratory sounds were normal, with the addition of coarse muco-crepitus over base of right lung. The action of the heart was remarkably irregular, and at the rate of 150 in the minute; impulse strong and labouring, thus contrasting very strikingly with the radial pulse. Both cardiac sounds were clear and ringing; they were extensively transmitted over the front of the chest, especially in the course of the aorta, and were unattended with murmur.

On the following day the pulse was perceptible at the wrist, but less distinct on the right than on the left side; it was 84 in the minute. Ordered a tonic, consisting of iron and chloric ether, with infusion of quassia.

June 10th. Œdema of lower limbs much increased; orthopnoea; congestion of base of left lung. To have, every sixth hour, a draught consisting of tincture of digitalis, ℥v; spirit of

juniper, 3j; and camphor water, 3j. To be dry-cupped over left base.

On the 15th, a wiffing systolic murmur was heard at the left apex; it was not audible elsewhere. The second sound was intensified in the pulmonary artery. The base of the left lung was dull, and yielded feeble respiratory sound with crepitus. There had been slight hæmoptysis in the course of the preceding night. The legs were somewhat swollen, and the patient complained of a stabbing pain below the left nipple. To have an opiate plaster applied there.

On the 19th, a grating murmur was heard at the apex; it preceded the first sound immediately, occupying the latter part of the diastolic pause. Four days previously I had inferred, from the general condition of the patient, the *probable* existence of mitral narrowing, and mentioned my impression to the clinical class; but I did not venture upon a positive diagnosis in the absence of the pathognomonic murmur, which was only heard for the first time on the 19th. On that day an affirmative diagnosis of mitral contraction was made.

On the 21st, the pulse-rate being 114, the respiration 30, and the action of the heart very irregular, a systolic murmur was alone heard at the apex. On the 22nd, this was no longer audible, whilst a harsh presystolic murmur was heard at intervals. The heart acted irregularly, both in regard to force and rhythm; it contracted spasmodically and with much energy several times in rapid succession, every three or four minutes; in the intervals it acted feebly. Coincidentally with the strong contractions, the presystolic murmur was heard; but it was inaudible during the period of feeble action.

On the 23rd it was very distinct, and on the 25th it was again all but suppressed.

27th. Copious expectoration of dark unmixed blood; presystolic murmur faintly audible. To have ℥x of dilute sulphuric acid in 3j of water every third hour.

28th. Great respiratory distress, and evidence of effusion on both sides of the chest; pulse 162; hæmoptysis continued. She died on the following day.

The body was examined by my resident clinical clerk, Mr.

O'Hanlon, eight hours after death. Both pleuræ and the pericardium contained a large quantity of serum. The left lung presented, on its anterior edge, a nodule of sanguineous infarction, as large as a walnut, and on its outer surface a small mass of cretified tubercle. There were, in the inferior lobe of the right lung, several large collections of solid and extravasated blood, and in the superior lobe numerous but smaller infarctions, so that the surface of section presented a mottled appearance.

The heart weighed twelve ounces and three-quarters. There was a good deal of fat upon its surface, especially around the roots of the great vessels. The right auricle contained some dark blood-clot; the right ventricle was slightly dilated but much thickened, and the tricuspid orifice so much dilated as to permit the passage of four fingers placed in juxtaposition. Entangled in the columns of the anterior wall of the ventricle was a large flake of solid yellow fibrin, whence it extended into the pulmonary artery for a distance of two inches.

The left auricle was much dilated and hypertrophied; the appendix was thickened in a high degree, and the orifices of the pulmonary veins were greatly dilated; the superior and left admitting the index and middle fingers. The mitral orifice barely admitted the point of the index finger passed from the auricle. It presented the appearance of a narrow slit, with thick, smooth, and well rounded edges. Both segments of the valve had become rigid; the anterior and right, thicker than the posterior and left segment, presented a smooth but uneven surface towards the ventricle, quite to its root at the aortic opening; both it and the attached tendinous chords were white and glistening, and one of the latter, which was connected with the anterior fleshy column, was as thick as the little finger. Both segments, though thick and firm, were flexible, and fell together so as to close the opening, but not perfectly. When the ventricle was filled with water, the aorta having been compressed with the fingers, there was a slight leakage into the auricle, owing to the maladaptation of the edges of the valves. The left ventricle was normal. The aortic valves were healthy and competent. The aorta was dilated, and in the ascending and transverse portions of the arch it was somewhat patchy on the internal surface. The liver exhibited the effects

of tight lacing in early life ; it was contracted in its transverse diameter, and elongated vertically ; on its anterior surface there was a deep horizontal sulcus, corresponding to the edge of the united costal cartilages, the adjacent portion of the capsule being thick and opaque.* The suppression of the characteristic murmur of mitral narrowing for a period of eighteen days, and its subsequent unsteadiness, demand special notice. The extreme weakness of the patient during this entire period, affords, in my judgment, a rational explanation of the circumstance. This view receives support from the observation made on the 22nd of June ; namely, that the murmur was heard during the period of vigorous, though spasmodic action of the heart, but was inaudible in the intervals, when the heart acted feebly.

CASE CX.—Rheumatism, and Hepatic Disease ; Dyspnœa, Hæmoptysis, and Œdema ; Congestion of both Lungs, and Cough ; Pulse Small but Regular ; Increase of Precordial Dulness ; Presystolic and Systolic Murmur at the Apex ; Second Sound Double ; Pregnancy at the Eighth Month ; Premature Confinement ; Death. Autopsy : Enlargement of the Heart by Hypertrophy and Dilatation of Left Ventricle ; Thrombosis of Right Chambers ; Partial Stenosis of Tricuspid Opening, and Thickening of Tricuspid Valves ; Great Dilatation, and Partial Hypertrophy of Left Auricle ; Stenosis of Mitral Orifice by Cohesion of Valves ; Calcification of Auricular Surface of Mitral Valves and Adjacent Endocardium ; Dilatation and Hypertrophy of Left Ventricle ; Stenosis of Aorta.

Sarah M., aged twenty-six years, married, and in the eighth month of pregnancy, was admitted July 20th, 1869. She was the wife of a soldier, and had spent three years in India. Whilst there she had rheumatic pains and "liver complaint," the latter, however, unaccompanied with jaundice.

Eight months prior to admittance, she began to suffer from shortness of breath ; five months later, she repeatedly spat blood, and three weeks afterwards her feet became swollen.

State on admission : Feet, legs, and thighs greatly swollen,

* See *Medical Press and Circular*, July 3rd, 1866.

but pale. Pulse 96, small, regular, and equal at both wrists; loud and dry cough; both lungs congested posteriorly, but the right in a greater degree than the left. Respiration 36, greatly embarrassed, and gasping; face pale, but with a malar blush, and congestion of lips. Urine scanty, 1·017 in sp. gr., and free from albumen. Precordial dulness was somewhat increased in extent. At the point of apex-pulsation, which was in the nipple-line and fifth intercostal space, a loud presystolic murmur was heard, and immediately succeeding it, a soft systolic murmur. At the base, a systolic murmur was heard faintly, and by transmission. The second sound was double, and heard all over the precordium.

The pulsation of the foetal heart was detected near the umbilicus; it was slow, exceeding the rate of the maternal heart in a slight degree only.

The chest was dry-cupped and poulticed, an aperient was given, and subsequently an active and stimulant diuretic.

She was prematurely confined of a still-born child on the 21st, after a labour of ten hours' duration. Slight uterine tenderness, which was accompanied by a fetid discharge from the vagina, was detected on the 24th. The breathing was much relieved by the evacuation of the uterus, but the cardiac signs were as previously noted.

On the following day the extremities were cold, and she spat some blood; in the evening she began to sink, and at 5 P.M. on the 26th, she died.

The body was carefully examined, with the following result: The heart was very large, and weighed nineteen and a-half ounces; there was a good deal of fat upon its surface, and, owing to hypertrophy of the left ventricle, the apex was magnified and blunted.

The lining membrane of the aorta, pulmonary artery, and pulmonary veins, was hyperæmic, and of a deep crimson tint. The right chambers contained a large decolorized thrombus, which was engrained into the pectinate wall of the auricle, passed thence into the ventricle, where it was entangled by a number of filaments with the tricuspid valve and the columnæ carneæ, and from the ventricle it extended by a large cylinder into the pulmonary

artery. The thrombus was hollow, constituting a thick and smooth lining to the chambers, and through it the circulation must have been carried on during the last hours of life. The right auriculo-ventricular orifice was contracted, admitting only the tips of two fingers; the segments of the tricuspid valve were thick, but apparently not incompetent. The right auricle and ventricle were normal as to capacity and thickness of walls. The left auricle was greatly dilated, and in a slight degree thickened; it was full of dark clotted blood. The lining membrane of the auricle, to the extent of half an inch around the mitral orifice, was rough and spiculated by deposition of a nearly uniform layer of calcareous matter.

The segments of the mitral valve were greatly thickened, and agglutinated together so as to form a circular curtain, rough and cretified on the auricular surface, but uniformly smooth and free from lime-salts on the ventricular side. In the centre of this curtain, which was rigid and immovable, there was an opening which was transversely oval, smooth on the edges, and barely large enough to admit the point of the index finger. The connected tendinous chords were much thickened, and the papillary muscles and the fleshy columns generally were hypertrophied. The left ventricle was dilated and greatly hypertrophied. The valves of the aorta were thick, but pliant, smooth, and competent, and the orifice was reduced to the diameter of the point of the little finger.

The quantity of blood which, in this case, passed into the aorta at each systole of the left ventricle, must have been very small, owing to the twofold lesion of mitral obstruction and incompetency. Hence, the extreme contraction of the aortic orifice, and notwithstanding this condition, the absence of a murmur of aortic obstruction. The opposite characters of the two murmurs heard at the apex of the heart, the harsh and grating quality of the presystolic, and the soft and blowing character of the systolic murmur, were alike explained by the conditions of the corresponding surfaces of the mitral valve. Hypertrophy and dilatation of the left ventricle were due, primarily and mainly, to inadequacy of the mitral valve.

CASE CXI.—*No History of Rheumatism ; no Hæmoptysis ; Recurrent Bronchitis ; Pallor and Debility ; Rapid but Regular Pulse ; Tumultuous Action of Heart, and Projection of Precordium ; Presystolic Fremitus and Murmur ; Postsystolic Musical Note ; Improvement ; Sudden Right Hemiplegia and Aphasia ; Death. Autopsy : Plugging of Left Anterior and Middle Cerebral Arteries ; Enlargement of Heart ; Thrombosis of Right Chambers and Pulmonary Artery ; Dilatation of Right Ventricle ; Dilatation and Hypertrophy of Left Auricle and Ventricle ; Extreme Stenosis of Mitral Orifice by Cohesion and Thickening of Mitral Valves ; Attachment of a Movable Thrombus to Anterior Segment of Mitral Valve ; Narrowing of Aorta, and Thickening and Inadequacy of Aortic Valves.*

Robert F., aged seven years, an inmate of a reformatory, was admitted into the Mater Misericordiæ Hospital on the 5th of November, 1872. He had not had rheumatism, nor had he spat blood. When admitted he was suffering from bronchitis. On examining him the following day, I found his condition to be as follows: He was pale and emaciated, and evidently had been indifferently nourished; he coughed, was feverish, and the pulse was rapid (120), but regular. The precordium projected considerably, especially on the left side, and the heart pulsated tumultuously. The apex-pulsation was felt in the fifth intercostal space, half an inch inside the nipple-line. At this point a presystolic fremitus was readily detected, and from this sign, without proceeding further with the examination, the diagnosis of mitral narrowing was at once made. A presystolic murmur likewise existed at the apex.

Both sounds of the heart were remarkably clear and sharp; but succeeding the first sound, and appended to it, was a musical note, which was likewise distinctly audible in the left axilla and back.

He improved under ordinary treatment, the bronchitis was less troublesome, and in the course of four or five days he was allowed to leave his bed and walk about the ward. By some

misadventure he contracted fresh cold, and had another attack of bronchitis which was more severe than the former.

He was confined to bed on the 18th and 19th, and exhibited greater debility and respiratory distress.

On the evening of the 19th his condition had undergone some improvement, and he declared that he felt better. He was seen by the Sister in attendance at 10 and 11 o'clock, respectively, when he seemed better. On the following morning he was found to be quite paralyzed on the right side, and unable to utter a word.

When I saw him at 11 o'clock on the 20th, he was completely paralyzed as to motion in the right arm and leg; but sensibility was in these limbs rather exalted. The features were slightly drawn to the left; the pupils were normal and equal, and there was no strabismus. He perfectly understood what was said to him, and in the effort to reply, his features were drawn more to the left side; he could not utter a word save "yes" and "no," and even these were expressed very indistinctly.

The pulse was rapid, the skin was hot and dry, and loud bubbling crepitation was heard all over the chest. He either could not or would not protrude his tongue; but deglutition, as regarded liquids, was perfect. The tactile and acoustic signs at the precordium were those previously noted.

He died on the evening of the 20th, evidently from accumulation of mucus in the air-passages, and the body was carefully examined by Dr. Nixon on the following day.

The brain was large, and nowhere softened; the arteries at the base were empty, except the left anterior and middle cerebral, both of which were plugged to distension with small and unequal sized particles of solid fibrin. The terminal portion of the carotid, at the proximal side of the obstruction, was empty; but the branches arising from both the infarcted vessels, at the distal side, were full of dark liquid blood.

The bronchial tubes throughout were congested, and contained much blood-stained frothy mucus. The heart was somewhat enlarged; it was globular in figure; the right chambers contained some dark soft coagulum, and a few detached masses of fibrin;

the former extended into the pulmonary artery, and was there coated with a film of fibrin.

The right ventricle was dilated and thinned; the left auricle and ventricle were dilated and hypertrophied, and the cavity of the left ventricle was rounded.

The aorta, including the orifice, was reduced in calibre, and the aortic valves were thick and puckered, but pliant; they were inadequate to close the orifice, as shown by the water-test.

The mitral orifice was constricted in a high degree; it admitted the point of the index finger with some difficulty; its figure was that of a button-hole, and it was bounded by the valve-segments, which were smooth and thick on the edges, being elsewhere fused into an elliptical curtain. To the anterior and right portion of this curtain a mass of solid fibrin was attached. This body was of the size and shape of a musket ball, but rough and shreddy on the surface; it involved, in its substance, about one-half the depth of the valve, and projected into the opening in such a way that it must have been wafted across the orifice of the aorta by the stream of blood entering the ventricle from the auricle. The emboli found in the cerebral vessels were, no doubt, derived from the jagged surface of this mass of fibrin by attrition of the blood-stream.

I cannot, with equal confidence, attribute to the vibration of this body in the mitral orifice at the acme of ventricular systole, the musical note of postsystolic rhythm which was heard at the apex and in the left axilla and back on the 5th of November, and repeatedly afterwards. My difficulty arises from the consideration that such a mode of origin would necessarily imply, either a murmur of mitral regurgitation, which did not exist, or, assuming that the body prevented reflux by acting as a ball-valve, the impossibility of its freely vibrating whilst so acting.

The case illustrates, firstly, the not unfrequent absence of a record of rheumatism in the history of mitral stenosis, even in the child; secondly, the precipitation of fibrin from the blood whilst still in circulation, owing to the twofold cause of partial stasis, and obstruction by a rough or uneven obstacle; and lastly, the detachment by the circulating blood, of detritus from the surface

of the thrombus so formed, its impaction in the left middle and anterior cerebral arteries, by which the left motor centre and Broca's region of speech were deprived of arterial blood, and right hemiplegia and aphasia were produced.*

CASE CXII.—*Acute Rheumatism ; Strain of Right Arm, and Aneurism by Rupture of the Ulnar Artery ; Palpitation ; Sudden and Complete Right Hemiplegia, with Partial Aphasia ; Visible and Collapsing Pulse ; Pulsating Tumor on Front of Right Forearm ; Dyspnœa and Hæmoptysis ; Œdema ; Double Basic, and Apex-Presystolic Murmur ; Death. Autopsy : Clot in Left Hemisphere of Brain ; Great Enlargement of Heart ; Disorganization of Aortic Valves ; Contraction of Mitral Orifice, with Deposit of Beaded Lymph upon Auricular Surface of Mitral Valve ; Fatty Degeneration of Heart, and Fatty Condition of Liver ; Large Aneurism of Right Ulnar Artery.*

Michael C., aged twenty-three years, a wire-worker, was admitted under Dr. Nixon's care, in September, 1872. By Dr. Nixon's kind permission I examined the man and took notes of his case.

The patient's habits had been temperate, but he had had three attacks of acute rheumatism. Twelve months previously, after working for an entire day with a pair of blunt pliers, which required powerful grasping pressure with the hand, he felt numbness and tingling in the three inner fingers of the right hand, which was the one he had been using. Shortly afterwards he noticed, on the inner portion of the anterior aspect of the right forearm, a swelling, which "panted"; this swelling continued to be of the original dimensions up to the date of his admittance.

He had been, for some time, subject to palpitation on exerting himself; and six weeks prior to admission he suddenly experienced giddiness, and, at the same time, lost the use of his right

* See *Proceedings of Pathological Society*, vol. v., part ii., new series, p. 118.

arm and leg; his speech was likewise, but only partially, affected, but feeling remained perfect in the paralyzed limbs.

When I saw the man, his right arm was quite powerless; his right leg was likewise paralyzed, but in a less degree; whilst in both, sensibility was unimpaired. He was pale; the features were slightly drawn to the left; articulation was thick and slow, but the tongue was protruded in the axial line. The pulse was about 84, visible and collapsing, but it was regular and equal on both sides. A swelling existed on the ulnar portion of the front of the right forearm, an inch and a-half below the internal condyle of the humerus; it yielded a pulsation, which was full and expansile, and synchronous with the radial pulse. Pressure on the brachial artery above the condyle stopped the pulsation in the tumor, and direct pressure entirely removed the swelling. If, whilst the hand remained on the tumor, pressure were suddenly withdrawn from the artery above, pulsation was at once restored in the former, and a feeling of sudden influx, accompanied by thrill, was communicated to the hand.

At midsternum, and likewise at the apex, but by transmission, two murmurs were heard. Of these, one was faint, and systolic in time, whilst the other was loud and whiffing, and *succeeded* a nearly normal second sound; the former alone was audible in the arteries of the neck.

On again visiting the man on the 25th of October, I found that he had regained all but perfect use of the right limbs, and entirely recovered the power of articulation. The ulnar aneurism had undergone slight enlargement. The physical signs were as previously described, with this addition, that a faint presystolic murmur now existed at the left apex. He was shortly afterwards discharged.

The man was readmitted for Dr. Nixon, on the 25th of January, 1873, and I was again favoured with the opportunity of examining him. He was then greatly changed; the face and neck were puffed and congested, respiration was greatly embarrassed, and the feet and legs were swollen.

The ulnar aneurism seemed to me unchanged, but the aortic murmurs were both much more harsh; the apex presystolic murmur was more distinct. He had hæmoptysis accompanied

by great dyspnoea on the 3rd of February, and he died on the 4th.

Autopsy : The heart was greatly enlarged, and weighed twenty-six ounces ; the right ventricle was thin and dilated ; it exhibited dun mottling on its internal surface, and the portions so changed in colour were found to have undergone complete fatty transformation. The tricuspid and pulmonic orifices and valves were normal. The left auricle was dilated and somewhat thickened ; its lining membrane was opaque. The mitral opening was narrowed to the size of a florin piece, and the mitral valves were studded, on the auricular aspect and near the attached margin, with a series of fibrinous nodules, as large as millet seeds, and arranged linearly ; the valves were otherwise normal, and were competent. The left ventricle was greatly dilated and thickened ; it was likewise mottled of a dusky yellow internally, and where these spots existed, it was structurally identical with the discoloured portions of the right ventricle.

The aortic valves were utterly disorganized ; they were rolled up into coils, were rough and friable, and beaded with nodules of fibrin.

The aorta was slightly atheromatous. A large blood-clot, nearly deprived of colour, was found in the medullary substance of the left cerebral hemisphere ; it had descended into the lateral ventricle, where it rested on the outer side of the corpus striatum. A small branch of the left middle cerebral artery had been closed, and converted into a fibrinous cord.

The liver cells contained a good deal of oil, and many of the muscular fibres of the heart were in an advanced state of fatty change. An aneurism existed upon the right ulnar artery, one inch and a-half below the bifurcation of the brachial.

In reference to the condition of the mitral orifice, Dr. Nixon made the following judicious remarks when reporting the case and exhibiting the diseased parts before the Pathological Society of Dublin :* " The beads of lymph found on the auricular surfaces of the mitral valves were certainly of themselves sufficient in amount to produce obstruction to the passage of the blood into

* See *Proceedings*, vol. v., part ii., new series, page 137.

the ventricle; and I think from the dilated condition of the latter, the large reflux of blood from the aorta must have brought the mitral curtains towards each other, thus exposing their roughened auricular surfaces to the passage of the blood, and thus producing the obstructive murmur."

No doubt, the premature distention of the ventricle by the sudden entrance of a large volume of blood from the aorta was, by depressing the valves, a contributory, though only a secondary, cause of the presystolic murmur, and must have intensified it considerably in the last days of the man's life.

CASE CXIII.—*Repeated Attacks of Rheumatism; Intemperate Habits; Hæmoptysis; Œdema; Hepatic Engorgement and Tenderness; Quick and Weak Pulse; Outward Displacement of Apex; a Presystolic and a Systolic Murmur at Apex-Point; Harsh Systolic Murmur at Right Base, and Transmitted into the Carotid Arteries; a Superficial Systolic Grating at Left Base; Second Sound Loud and Sharp; Sudden Loss of Power in Right Hand and Forearm, and soon afterwards, Sudden Death. Autopsy: Congestion of Lungs and Liver; Adhesion of Pericardium, and Enlargement of Heart, with a Cheese-like Mass Imbedded in its Anterior Surface; Thrombosis of Right Chambers; Dilatation of Right Ventricle; Dilatation and Hypertrophy of Left Auricle and Ventricle; Extreme Narrowing of Mitral Orifice, and Calcareous Conversion of Mitral Valves; Calcareous Change, but without Incompetency, of Aortic Valves.*

Thomas W., aged thirty-six years, a blacksmith, of very intemperate habits, was received into hospital, May 30th, 1873. He had had several attacks of rheumatism, the first at the age of sixteen years. His breathing had been short for the last twelve years. One year prior to admission he began to spit blood, and hæmoptysis had recurred at intervals since that date. His feet had been swollen for the last month.

State on admittance: The back was arched, the chest narrow, and the sternum prominent. Pulse very weak but regular, and 108. Throbbing of the carotids, and œdema of the lower limbs.

He complained of pain in the left side of the chest from the clavicle downwards, and spat blood daily in large quantity. Breathing not much embarrassed; occasional cough; features sunken. The kidneys acted indifferently; the urine was loaded with lithates, but was otherwise normal; the liver was engorged, and tender to pressure, and the bowels were inflated; the impulse of the heart was feeble; the apex-point was in the nipple-line, and there a presystolic and a loud systolic murmur were heard in unbroken continuity. The initial portion, corresponding to the presystolic constituent of this prolonged and compound murmur, was readily distinguished from the latter or systolic portion, by its being harsh and emphasized, and by its obviously preceding the apex-pulsation.

At the right base, a rough systolic murmur existed; it was faintly transmitted into the arteries of the neck. At the left base, a coarse and superficial rub of systolic rhythm was heard. The second sound at the base was loud and sharp, but no diastolic murmur existed.

On the night of the 2nd of June, he suddenly lost the use of the right hand and forearm, which then became gradually swollen; yet no difference between the radial pulses could be perceived. There was total loss of rest. To have six leeches at the epigastrium, and ℞v of Battley's sedative at night. His condition underwent no marked improvement, and he died suddenly on the 12th.

Autopsy, twelve hours afterwards: The lungs were voluminous, and engorged with blood; the bases were solid, and dark in section. The anterior surface of the heart, and the corresponding portion of the parietal pericardium, were roughened by effused lymph, and the opposed surfaces were connected by a few long bands of fibrin. In the anterior surface of the right ventricle, and surrounded by adhesion, a cheese-like mass, about as large as a pea, was imbedded. The heart weighed twenty and a-quarter ounces; there was a good deal of fat on its surface, chiefly in the grooves. The aorta was of normal size, and free from disease. The right chambers contained a mass of solid fibrin, which extended into the pulmonary artery; the right ventricle was dilated and thinned; the valves on the right side of the heart were

healthy. The left auricle was greatly dilated and hypertrophied, and its lining membrane was thick and opaque; the left ventricle was dilated and much thickened. The mitral orifice was reduced to the diameter of the point of the little finger; it was of a button-hole shape, and its valves were shrunk and converted into a calcareous but friable mass, which was remarkably rough on the surface; the anterior and right segment was more disorganized than the posterior and left.

Both the anterior segments of the aortic valve were converted into rugged, calcareous masses, which projected into the orifice; but the posterior segment was soft and pliant, and so much expanded that it overlapped the diseased segments, covering the axial passage left between them so as to prevent regurgitation. The orifices of the coronary arteries, and the vessels themselves, were unaltered. The liver was large and congested; the kidneys were healthy.

CASE CXIV.—*Rheumatism; Intemperance; Hæmoptysis and Dyspnoea; Anasarca and Ascites; Albuminuria; Weak Pulse; Jugular Pulsation; Systolic Murmur, accompanied by a Musical Note, at the Apex of the Heart and over the Body of the Ventricle; Feet Punctured, and Rapid Dispersion of Dropsical Effusion; Gangrene of Feet and Legs; Asystole; Death. Autopsy: Serous Effusion into the Peritoneum; Enlargement of the Liver and Spleen; Kidneys Enlarged, and in Transition Stage to Atrophy; Lungs Emphysematous; Heart Enlarged, and Adherent to Pericardium; Right Chambers Hypertrophied and Dilated; Tricuspid Orifice Dilated, and Valves Thickened; Left Chambers Dilated and Hypertrophied; Mitral Orifice greatly Contracted, and Valves Calcified; Aorta Dilated and Atheromatous; Pulmonary Artery Dilated.*

Matthew N., aged fifty-eight years, a sailor, was received into hospital, August 25th, 1874. At the age of seventeen he had rheumatic fever which lasted three months; had been a hard drinker, and had repeatedly spat blood. Two years prior to date of admittance he had an attack of bronchitis, and then for the first time noticed his breathing to be very short. His feet had

become swollen within the previous six weeks, and when he was admitted the following was his condition. The lower limbs were greatly swollen; there was some liquid in the peritoneum; respiration was embarrassed; pulse 108, and very weak, but regular; urine scanty, 1·025 in sp. gr., acid, and containing some albumen and blood. The external jugular veins were full, but not distended; they pulsated synchronously with ventricular systole. There was comparative dulness with crepitation over the base of the right lung. The area of precordial dulness was contracted, owing to an emphysematous condition of the edges of the lungs. The apex-pulsation could not be distinctly felt; but, two inches below the nipple and in the fifth intercostal space, a systolic bellows-murmur of high pitch, and terminated by a sharp musical note, was audible. This murmur was still more distinctly heard in the fourth intercostal space, midway between the nipple and the sternum, and it was faintly audible for a distance of two inches upwards and to the right; it could not be heard in the axilla or in the back. In the course of a few days it became necessary to puncture the feet in order to avert gangrene. A copious flow of serum from the punctures took place. Diarrhœa, accompanied by diuresis, set in, and under the combined influence of this twofold drain, the œdema of the lower limbs and the ascites were entirely dispersed in less than forty-eight hours. The pulse was now 96, very weak, slightly irregular and faltering. The following tracing (Fig. LX.), taken by Dr. Nixon, exhibits these characters.



FIG. LX.

Mitral constriction and tricuspid regurgitation.

Matthew N. Pulse 96. September, 1874.

For a week subsequently the diarrhœa continued at the rate of about twenty motions in the twenty-four hours, and the discharge from the punctures was kept up. During this period, de-

spite the frequent calls to the night-chair, the poor man experienced the greatest relief; he could sleep in the recumbent posture, and was quite free from suffering. He had been taking, thrice daily, ℥v of tincture of digitalis with ℥x of tincture of the perchloride of iron; this was now suspended.

On the 15th of September, diarrhœa having for some days ceased, anasarca and ascites began to re-appear. A repetition of puncturing soon afterwards became necessary; but, despite this measure, gangrene of the feet and legs followed, and after three days of great suffering, during which no pulse could be felt at the wrist, the patient died on the 2nd of October. The body was examined on the following day.

Abdomen: The peritoneum contained several quarts of serum; the liver was enlarged, and in an early stage of cirrhosis; it weighed four pounds seven ounces, was rough on the surface, firm in texture, and its interstitial fibrous tissue was hypertrophied; the gall-bladder was distended with bile. The spleen weighed eleven ounces, was firm, and its capsule was thickened. The right kidney weighed seven ounces, and the left kidney six ounces; they were both firm in texture, light-coloured in section, and the tubes were loaded with detached epithelium; they were manifestly in the transition-stage, from hypertrophy to atrophy, of the "large white kidney."

Peyer's patches were somewhat magnified, but there was no ulceration or hyperæmia of the bowels.

Thorax: The lungs were voluminous and emphysematous. The heart was enlarged, globular in figure, and firmly attached to the pericardium by old adhesions on the anterior and posterior surface; it weighed nineteen ounces. Both right chambers were hypertrophied and dilated; they contained a good deal of blood-clot. The tricuspid orifice was greatly magnified, and the tricuspid valves were somewhat thick, opaque, and fringed with red; they were incompetent owing to dilatation of the orifice. The left auricle and ventricle were dilated and hypertrophied, and the orifices of the pulmonary veins were much enlarged. The mitral opening was greatly contracted; viewed from the ventricle, it was of the size and figure of a coat button-hole, somewhat rounded at the anterior extremity, and larger

than at the posterior. On the auricular aspect it presented a crescentic figure, convex anteriorly. The valve-segments were thick and perfectly rigid; they were universally calcified, but, being invested by the endocardium, they were smooth at all points except near the edge and on the auricular surface, where they were slightly spiculated. The attached tendinous chords were attenuated, and quite free from calcific change. The aorta was dilated above the valves and throughout the arch; it was in an early stage of atheroma. The aortic valves were healthy and competent, as were likewise those of the pulmonary artery. The last mentioned vessel was dilated, but its valves were competent. The muscular structure of the heart was healthy.

The chest had been repeatedly examined, and the diagnosis arrived at was, Mitral and tricuspid regurgitation, most probably from cartilaginous thickening of the valves, with fibroid transformation of the papillary muscles and inner layer of the ventricular walls. Presystolic murmur was not at any time detected; and, as the heart was carefully and almost daily examined during the thirty-nine days of the man's residence in hospital, I am satisfied such could not have existed. During the entire of this period the pulse was weak and small, but not faltering, or irregular.

The case, then, illustrates protracted suspension of presystolic murmur. This, I believe to have been due to debility of the left auricle, arising from general weakness.

CASE. CXV.—History of Cardiac Disease of Long Standing; Repeated Hæmoptysis; Orthopnoea; General Dropsy and Cyanosis; Increased Area of Precordial Dulness; Presystolic Murmur at the Apex, and also at the Left Margin of the Sternum; Gangrene of the Feet and Legs; Diagnosis of Mitral and Tricuspid Narrowing; Death. Contraction of both Auriculo-Ventricular Orifices.

Margaret M., aged twenty-five years, unmarried, was sent to me by Dr. Finegan, medical officer to the Finglas Dispensary, and admitted into the Mater Misericordiæ Hospital on the 11th of March, 1874. She had had measles of a bad type at the age

of sixteen. During convalescence, she caught cold from premature exposure, and was never subsequently in good health; her breathing became short, and she was subject to dizziness and palpitation.

Five years prior to admittance she spat some blood, and she had repeatedly spat blood subsequently. Menstruation ceased in December of the preceding year. Three weeks before admission and coming under my notice, her feet began to swell, and there was, at the same time, a decrease in the renal secretion. The following was her actual condition on the 11th of March. There was venous congestion of the face, and great turgescence of the veins of the neck; general lividity and depressed temperature of the surface; great cedema of the lower extremities; oppression of breathing, and orthopnoea. There was constant teasing cough, with slight frothy expectoration. The pulse was rapid, and so feeble that it could not be counted at the wrist. There was evidence of cedema of the bases of both lungs, and of effusion into the peritoneum. Precordial dulness was extended towards the right side; the impulse of the heart was strong and rather heaving, and the apex pulsated to the left of the nipple-line. Over the apex of the heart a well pronounced presystolic murmur of the ordinary character was heard; it was rough, but not remarkably so, and it extended quite up to the first sound.

In addition, there was audible to the left of the sternum, over the inner portion of the fifth costal cartilage and the corresponding part of the fourth intercostal space, a murmur of the same rhythm, but much more harsh than the former, and extending farther backwards into the long pause. At a point intermediate to the seats of these two murmurs, neither was distinctly audible.

The last mentioned circumstance I considered of so much significance, that upon it mainly, I ventured to make the diagnosis of obstruction at both auriculo-ventricular orifices. I repeatedly stated this opinion in presence of the clinical class, and I also mentioned it to my colleague Dr. Nixon whilst discussing the case with him. The poor woman's condition admitted of alleviation only. Gangrene of the feet and legs occurred, and she

died exhausted on the 14th of March, four days after her admission.

On examination of the body, the lungs were found to be congested and œdematous. There was some liquid in the pericardium. The heart was somewhat globular in figure, and weighed eleven ounces. The right auriculo-ventricular orifice was so reduced in diameter as to admit only the point of the middle finger; the tricuspid valves were slightly thickened, and united at the angles. The right auricle was much dilated and hypertrophied. The right ventricle was not dilated; it contained a good deal of blood-clot and decolorized fibrin, part of which was entangled in the tricuspid opening. The mitral orifice was still more contracted than the tricuspid, admitting only the point of the index finger.

The left auricle was thickened and dilated in a high degree, and the orifices of the pulmonary veins were dilated.

The left ventricle was not dilated or hypertrophied; it was remarkably thin at the apex, near the septum.

The point of greatest diagnostic importance in this case was the existence of two centres of presystolic murmur; one occupying the usual situation at the apex of the heart, and the other corresponding to the sternal end of the fifth costal cartilage and fourth intercostal space on the left side. Around these points, and within a circle of about two inches and a-half in diameter, the murmurs were respectively audible; but between and at an equal distance from them, there was a space where neither murmur could be distinctly heard.

This constituted the principal element on which the differential diagnosis was based. Corroborative evidence was, however, afforded by the difference in quality and in length of the murmur heard at the two points mentioned, a difference which I incline to attribute to the entanglement of fibrin in the edges of the tricuspid valves; and also by the existence of general venous turgescence and cyanosis. This latter condition is so exceptional in cases of mitral constriction, that, if copious effusion into both pleuræ were not likewise present, I should regard it as presumptive evidence of the existence of the double lesion.

In none of the published cases of mitral and tricuspid narrow-

ing combined, was the second sound reported to have been accentuated or doubled; a circumstance to be attributed, in my judgment, to the fact that, as regarded obstruction in front, and defective supply from behind, the two ventricles were in a similar predicament. The contracted auriculo-ventricular orifices served, each, as a point of resistance to the opposite ventricle, and as a stop to the corresponding ventricle. Hence, the absence of unequal dilatation and hypertrophy of the ventricles, and of doubling and intensification of the second sound.*

CASE CXVI.—*Edema of Feet; Frequent and Copious Hæmoptysis; Rheumatic Pains; Congestion of Face, and Exophthalmia; Dyspnœa and Cough; Headache and Recurrent Syncope; Pain in Region of Heart extending down the Left Arm, and in both Scapular Regions; Congestion of Right Lung and of Liver; Heaving Impulse at Precordium; Apex-pulsation much to the Left of Nipple Line; Postdiastolic Murmur at Apex; Subsequent Development of Presystolic Murmur, which was twice afterwards Suppressed and Restored alternately; Fremitus; Accentuation of Second Sound; Patient Six Years under Observation. Diagnosis: Progressive, and now Extreme Mitral Stenosis; Great Dilatation of Right Ventricle, and Consequent Displacement of the Apex to the Left Side; Exophthalmic Goitre.*

Mary E. C., aged twenty-five years, a machine sewer, consulted me for the first time in the summer of 1868. Of this visit I have preserved no record beyond the date. Her second visit was made on the 30th of June, 1869. On the latter occasion, I learned that for the last two months the feet had exhibited slight cedema in the evening, and that she had spat some blood. Has teasing dry cough; breathing much oppressed; pulse 120, small, and regular; face florid, eyes prominent, and thyroid body somewhat enlarged, but no distention of the cervical veins. She has frequently suffered from headache, and has constant sharp pain in the left side below the nipple; cannot lie on that side,

* Case 61 (p. 710) completes the list of fifteen fatal cases of mitral stenosis in which the body was examined after death.

or sleep in the recumbent posture. Cardiac impulse heaving, and apex-pulsation half an inch outside the left nipple-line. The sounds of the heart were normal at the base, but at the apex a loud bellows-murmur of postdiastolic rhythm was heard. The second sound was accentuated in the pulmonary artery. To have ether and ammonia in camphor water.

October 5th. Pain very severe in the region of the heart, and extending down the left arm, as far as the elbow. There are fits of dyspnœa, even when she is at rest. Apex-pulsation is now felt one inch outside the nipple-line, and here, as previously, a loud postdiastolic murmur of blowing quality is heard. To have carbonate of ammonia, grs. iij, with tincture of digitalis and chloric ether, of each ℥v, thrice daily; grs. v of Dover's powder at night, and xxx drops of ozonic ether (Richardson) by inhalation, during paroxysms of dyspnœa.

November 24th. Has repeatedly spat blood since last report. Cough is now very troublesome; severe pain in region of heart; acoustic signs as previously noted. To have two leeches at seat of pain, and a cough mixture containing chlorodyne and morphia.

February 2nd, 1870. A fortnight since she threw up a large quantity of blood, after which she fainted, and was insensible for about an hour. One week later, the hæmoptysis and syncope recurred whilst she was at work, and on that occasion, according to the report of her companions, her face became congested. Pulse 110, and regular; feet somewhat swollen in the afternoon; cheeks congested and purple; pain at apex of heart, and orthopnoea. The cardiac signs were unaltered.

She was admitted into hospital on the 3rd of February, and remained under treatment about two months. During this period she coughed up large quantities of blood from time to time, and suffered from severe pain at the heart. The latter was always promptly relieved by leeching. The annexed tracing (Fig. LXI.) represents the character of the pulse at that date.



FIG. LXI.

Extreme mitral constriction.

Mary E. C. Pulse regular, but very feeble. March 24th, 1870.

October 31st. Several weeks ago, whilst at work, she was struck on the left side of the abdomen with great violence, by the handle of a machine, and was rendered incapable of resuming her labour for some time. Kidneys acting defectively; cough and dyspnoea; feet swollen at night. There has been occasionally a streak of blood in the sputa; she complains of pain at the seat of injury, and likewise at the precordium. At the point of apex-pulsation, which is now located one inch and a-half externally to the nipple-line, a faint presystolic, and a loud postdiastolic, murmur are audible; both murmurs may be likewise heard, but much less distinctly, midway between the nipple-line and the sternum; they are not audible at the base, where the sounds of the heart are normal. Pulse 96, regular, and moderately full. To have tincture of digitalis and chloric ether, of each ℥v, thrice daily.

October 19th, 1871. After an absence of nearly a year, this girl called on me to-day. She now suffers occasionally at night from pain of a burning character in both scapular regions, but more severe on the right than on the left side. Pulse 108, regular, and moderately strong; dyspnoea; congestion of face, and œdema of the ankles after the exertion of the day; dysuria. There is scarcely any cough, but the sputa are occasionally bloody. She can now sleep, and can pursue her avocation (machine sewing), but with some fatigue.

The apex-beat was one inch and a-half to the left of the nipple-line, and in this situation a postdiastolic murmur was alone heard. Cardiac sounds normal, and elsewhere unattended with murmur. To have tincture of digitalis with spirit of nitrous ether; two leeches to be applied in left infra-scapular region.

February 5th, 1872. Has had a febrile attack which confined her to bed for six weeks; no hæmoptysis for the last four months; great dyspnœa on ascending a flight of stairs, and likewise when she attempts to work the treadle of her machine; but after a short time the difficulty of breathing subsides, and she can then continue her work. Feet still swollen at night; face purple, and eyes prominent; congestion of right lung; pulse 84, small and regular. Apex-beat in fifth interspace, one inch and a-half outside the nipple-line, and felt over an area of one inch in diameter. In this situation, but strictly limited to it, a loud postdiastolic murmur is audible; it is continued from, but in no degree masks, the second sound, whilst it falls short of the first sound by a distinct interval of time. Both cardiac sounds are normal. The second sound is accentuated in the pulmonary artery.

April 29th. For last fortnight she has been suffering from irritability of stomach, and empty retching; has a feeling of weight at the pit of the stomach, and a "burning" sensation in both scapular regions. To have mild aperients, and Boudault's pepsine.

February 15th, 1873. Admitted again into hospital to-day. Is very weak; has cough and headache; face of a bright crimson tint; pulse 96, regular; no œdema. Apex-pulsation two inches external to nipple-line, and here there is a harsh and prolonged postdiastolic murmur, which is accompanied by fremitus; this is succeeded by a short period of silence; and then a harsh presystolic murmur and a normal first sound are heard in succession. No murmur is elsewhere audible. The second sound is not double or accentuated. On one occasion, whilst in hospital, she coughed up a teacupful of florid blood. She likewise complained of pain at the apex of the heart, which was relieved by the application of two leeches, followed by the administration of ℥xxx doses of the tincture, with 3j of the syrup, of the American wild cherry (*Prunus Virginiana*). The pulse, at this date, is represented in the tracing (Fig. LXII.).



FIG. LXII.

Mitral constriction. Mary E. C. June, 1873.

August 15th, 1874. Called on me to-day. Complains of pain beneath the left nipple, and in left back; has pain also in the joints of feet and hands. The face is purple; the breathing oppressed; she has a dry and teasing cough. Pulse 96, and regular. A loud and "harsh" postdiastolic murmur only exists at the apex of the heart.

The protracted period, more than six years, during which this case has been under my observation, and the great interest which attaches to it in regard to the progressive development of physical signs, may be accepted as my apology for publishing it, and at such length, in connexion with those in which the diagnosis was confirmed by autopsy.

CASE CXVII.—*Rheumatism; Dissipation; Hæmoptysis; Dyspœna and Œdema; Hepatic Congestion; Double Cardiac Impulse; Fremitus; Postdiastolic and Presystolic Murmur at Apex; Postdiastolic Murmur with Musical Note, at Base; Partial Left Hemiplegia and Left Hemiopia; Extensive Sub-retinal Extravasation. Diagnosis: Mitral Stenosis; Inadequacy of Aortic Valve from Laceration of one or more of the Segments; Capillary Embolism.*

Thomas K., aged twenty-four years, a man of very intemperate habits, and a float-driver by occupation, was received into hospital on the 16th of September, 1874. He had been previously admitted for rheumatic pains, from which he quickly recovered without having apparently sustained any serious detriment of health.

He had been recently leading a very irregular life; had drunk

a good deal of whiskey, and been exposed to hardship of various kinds. For nine days before admission, he had been expectorating florid blood, and for several nights he had not been able to lie down, owing to the difficulty of breathing which he experienced in the horizontal posture. When admitted, he was pale, bloated-looking, and very weak; he complained of a feeling of constriction at the sternum, and breathed with great difficulty; the feet were swollen. From a cursory examination made in the waiting room, I diagnosed mitral constriction and aortic regurgitation.

The man was placed under the care of Dr. Nixon, who, at my request, kindly transferred him to me on the following day.

The report on the 18th was as follows: He suffers from teasing cough with expectoration of blood-stained mucus; there is orthopnea, also cedema of the feet, visible pulsation of the carotids, and epigastric tenderness; pulse 96, full and abrupt, but not visible. The impulse of the heart was double. Apex-pulsation was felt half an inch inside the nipple-line, in the fifth interspace; at this point, in addition to the sounds of the heart, which were distinct and normal, two murmurs were audible; they were respectively postdiastolic and presystolic in rhythm. At the base, both sounds of the heart were sharp and abrupt, and the second was succeeded by a soft bellows-murmur which was accompanied by a musical note. This murmur was transmitted with great distinctness to the ensiform cartilage, and faintly to the right second costal cartilage, but not into the transverse portion of the arch, or the carotid arteries. No murmur was audible in the back. The base of the right lung was congested. The slightest movement of the body was followed by great palpitation and respiratory distress.

Leeches were applied at midsternum, the chest was dry-cupped posteriorly, and a mixture of chloric ether and tincture of digitalis was given. Fig. LXIII. shows the character of the pulse on the following day.

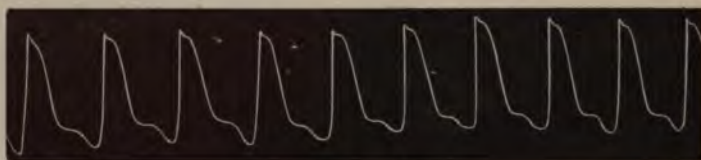


FIG. LXIII.

Mitral constriction and partial aortic regurgitation. Hypertrophy and dilatation of the left ventricle.

Thomas K. Pulse 112. September 19th, 1874.

On the night of the 20th he was suddenly attacked with headache, and with severe pain in the left side of the chest, extending down the left arm and leg. On the following morning he had lost vision in nearly one-half of the left retina; he could only see the upper portion of objects with that eye. The pupils were, however, normal and equal; hæmoptysis had continued. Fremitus was now detected at the apex, and in that situation only a single murmur was heard, which, however, extended over the entire long pause.

Three leeches were applied over the apex of heart, and gr. ij each of grey and of antimonial powder were given every second hour.

On the 23rd he was much quieter, having slept well the preceding night. The apex-murmur was now again double, *i. e.*, it consisted of a postdiastolic and a presystolic element, with an intervening period of silence; the latter was ingravescent; it was also more harsh than the former. The first sound at the base was accompanied by a faint murmur which was transmitted into the carotid arteries; whilst the second sound was accentuated in the aorta, and its satellite murmur was now audible in the transverse portion of the arch. The left eye underwent a careful examination with the ophthalmoscope to-day, by my colleagues, Mr. Hayes and Dr. Nixon. An extensive extravasation of blood was discovered in the fundus, by which the upper and outer portion of the retina was raised in large and irregular patches, radiating from the optic entrance.

The man can now sleep in the recumbent posture, is free from pain in the arm and leg, and from feeling of constriction in the chest. The œdema has been dispersed.

On the 28th he was allowed to be out of bed. For the preceding few days he had been taking, thrice daily, a draught

consisting of Hoffman's anodyne, 3ss ; Tincture of camphor and spirit of chloroform, of each, ℥xv. From this he appeared to derive great benefit.

There can, I think, be little doubt that capillary embolism, by migration of detritus from the aortic valve, took place on the night of the 20th, and was the cause of the pain in the left side of the body, and of the partial loss of vision by subretinal hæmorrhage in the left eye.

In cases of hemiplegia from embolism of the middle cerebral artery, so frequently witnessed in connexion with valvular disease on the left side of the heart, I entirely agree with Dr. Clifford Allbutt, and therefore differ from Niemeyer, in holding that loss of consciousness is of very rare occurrence. There is usually nothing more than temporary bewilderment from shock. The order of events which may be witnessed in cases of embolism of the central artery of the retina, is, according to Allbutt, the following, namely ; instantaneous loss of function, sudden emptying of retinal vessels, gradual œdema of portions of retina deprived of blood, hæmorrhage still later from collateral vessels, thickening of "adventitia," and fatty degeneration of retina with deposit of chlosterine.*

The subjoined tracing, though representing a combination of valvular disease identical as to kind with that shown in the preceding figure (LXIII.), contrasts strikingly with the latter by its want of regularity, and by the defective height and verticality of its upstroke. It represents, in short, incipient failure of the left ventricle from retrogressive tissue change.

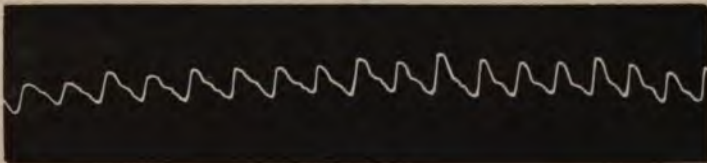


FIG. LXIV.

Mitral obstruction, and partial aortic regurgitation.

Peter F. (No. 61, Table XI.) September 23rd, 1873.

All the cases of mitral stenosis which came under my notice previously to 1875, are presented, in summary, in the succeeding Tables (XI. and XII.).

* *On the Use of the Ophthalmoscope*, 1871.

TABLE XI.—MITRAL STENOSIS.

No.	Name.	Age.	Hæmoptysis.	Dyspnea.	Edema.	Irregular or Intermitent Pulse.	Failure of Pulse.	Fremitus.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
1	Anne K. ..	28	1	1	1	1	1	1	1	1	1	1	1	Tricuspid inadequacy, vegetations on valve.	Funnel-shaped.	..
2	Ellen D. ..	30	1	1	1	1	1	1	1	1	1	1	1
3	Jane L. ..	33	1	1	1	1	1	1	1	1	1	1	1
4	Jane G. ..	34	1	1	1	1	1	1	1	1	1	1	1
5	Jane Q. ..	47	1	1	1	1	1	1	1	1	1	1	1
6	Mary S. ..	26	1	1	1	1	1	1	1	1	1	1	1
7	Jane McG. ..	50	1	1	1	1	1	1	1	1	1	1	1
8	John M. ..	50	1	1	1	1	1	1	1	1	1	1	1
9	John M. ..	50	1	1	1	1	1	1	1	1	1	1	1
10	Agnes B. ..	48	1	1	1	1	1	1	1	1	1	1	1
11	Mary M. ..	37	1	1	1	1	1	1	1	1	1	1	1
12	Robert McB. ..	14	1	1	1	1	1	1	1	1	1	1	1
13	Thomas N. ..	10	1	1	1	1	1	1	1	1	1	1	1
14	Mary F. ..	30	1	1	1	1	1	1	1	1	1	1	1
15	Mary B. ..	28	1	1	1	1	1	1	1	1	1	1	1
16	Patrick O. ..	40	1	1	1	1	1	1	1	1	1	1	1
17	Kate O'B. ..	34	1	1	1	1	1	1	1	1	1	1	1
18	Margaret B. ..	30	1	1	1	1	1	1	1	1	1	1	1
19	Patrick Q. ..	40	1	1	1	1	1	1	1	1	1	1	1
20	Bridget P. ..	24	1	1	1	1	1	1	1	1	1	1	1
21	Charles B. ..	17	1	1	1	1	1	1	1	1	1	1	1
22	Sarah M. ..	26	1	1	1	1	1	1	1	1	1	1	1
23	Bridget McG. ..	23	1	1	1	1	1	1	1	1	1	1	1
24	Mrs. E. ..	38	1	1	1	1	1	1	1	1	1	1	1

* Age not stated.

TABLE XI.—Continued.

No.	Name.	Age.	Hæmophysis.	Dyspnoea.	Oedema.	Irregular or Intermitent Pulse.	Failure of Pulse.	Fremittus.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
25	Lizzie H. . .	18	1	1	1	1	1	1	1	1	1	1	1	Aortic insufficiency.
26	Eliza H. . .	26	1	1	1	1	1	1	1	1	1	1	1	Post-systolic murmur covering nearly the entire aortic valve.
27	Mary O'S. . .	32	1	1	1	1	1	1	1	1	1	1	1
28	Anne R. . .	23	1	1	1	1	1	1	1	1	1	1	1
29	Sarah C. . .	46	1	1	1	1	1	1	1	1	1	1	1
30	John M. . .	22	1	1	1	1	1	1	1	1	1	1	1	Embolism of right and left hemiplegia.
31	John C. . .	22	1	1	1	1	1	1	1	1	1	1	1
32	Patrick McD. . .	23	1	1	1	1	1	1	1	1	1	1	1
33	Mrs. R. . .	30	1	1	1	1	1	1	1	1	1	1	1
34	Joseph F. . .	7	1	1	1	1	1	1	1	1	1	1	1
35	Mary M. . .	38	1	1	1	1	1	1	1	1	1	1	1
36	Miss C. . .	38	1	1	1	1	1	1	1	1	1	1	1
37	Miss H. . .	38	1	1	1	1	1	1	1	1	1	1	1
38	Mrs. R. . .	25	1	1	1	1	1	1	1	1	1	1	1	First sound of heart double.
39	Thomas D. . .	15	1	1	1	1	1	1	1	1	1	1	1
40	Emma McC. . .	26	1	1	1	1	1	1	1	1	1	1	1
41	Mary K. . .	26	1	1	1	1	1	1	1	1	1	1	1
42	Jane C. . .	24	1	1	1	1	1	1	1	1	1	1	1
43	Thomas M. . .	16	1	1	1	1	1	1	1	1	1	1	1
44	Robert F. . .	7	1	1	1	1	1	1	1	1	1	1	1	Embolism of right hemiplegia, and aphasia. Fatty degeneration of heart (see case 61, p. 710).
45	Mary A. . .	28	1	1	1	1	1	1	1	1	1	1	1

* Age not stated.

TABLE XI.—*Concluded.*

No.	Name.	Age.	Hemoptysis.	Dyspnea.	Edema.	Irregular or Inter- mittent Pulse.	Failure of Pulse.	Fremitus.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
46	Kate B. "	18	1	1	1	1	Had five different attacks of rheumatic fever.
47	Bridget McG.	28	1	1	1	1	1	1	Had three attacks of rheumatism; presystolic murmur became inaudible in state of debility.
48	Mary F. "	23	1	1	1	..	1	Embolism; right hemiplegia and aphasia.
49	Michael C.	23	1	1	1	1	Aortic obstruction and inadequacy.	Button-hole.	Right hemiplegia & partial aphasia; clot in left cerebral hemisphere.
50	Mary K. "	26	1	1	1	Embolism; right hemiplegia and aphasia; recovery.
51	Margaret C.	30	1	1	1
52	Michael F.	25	1	1	1	1	Tricuspid obstruction.
53	Mary McC.	40	1	1	1	1	1	1
54	Thomas W.	36	1	1	1	1	1
55	Patrick McD.	62	1	1	1	1	1	..	Aortic obstruction.
56	Daniel G.	27	1	1	1	1	1	1	Aortic inadequacy.
57	Michael L.	17	1	1	1	1
58	Theresa J.	12	1	1	1	1
59	Elizabeth B.	21	1	1	1	1
60	Bessie L.	59	1	1	1	1
61	Peter F.	48	1	1	1	1
62	Eliza B.	27	1	1	1	1
63	Eliza D.	17	1	1	1
63			35	24	17	14	9	34	31	26	16	18	13			

TABLE XII.—MITRAL STENOSIS (SUPPLEMENTARY).

No.	Name.	Age.	Hæmoptysis.	Dyspnoea.	Edema.	Irregular and Inter- mittent Pulse.	Failure of Pulse.	Pleuritis.	Rheumatism.	Double 2nd Sound.	Accent. 2nd Sound.	Systolic Murmur at Aper.	Autopsy.	Other valves affected.	Form of Mitral Stenosis.	Complications and Observations.
1	Mr. B.	42	1	Sudden amnesia of words, from which she re- covered.
2	Catherine P. ..	43	Neuralgia.
3	Mrs. McH. ..	40	Leg amputated for bad sore, by Mr. Hayes; pa- tient made a good recovery.
4	Eliza C. ..	50	1	Cold after measles in infancy.
5	Margaret M. ..	25	1	1	1	..	1	1	Tricus- pid ste- nosis.	Funnel- shaped in both.	Acute pericarditis. Acute pericarditis; loud metallic tinkle in sto- mach synchronous with impulse of heart.
6	Arthur C. ..	14	..	1	1	1	..	1	1	Presystolic murmur was suppressed for two days; after which it was again audible, and continued to be heard until she was discharged.
7	Thomas H. ..	25	Had seven different attacks of rheumatism. Three and a-half years previously I diagnosed mitral stenosis. Murmur inaudible for a period of two days. Localized pericardial friction-sound of systolic rhythm, at base.
8	Margaret McG.	25	1	1	..	1	..	Aortic inade- quacy.	..	Had three several attacks of rheumatism. Pulmonary emphysema and displacement of heart down, behind ensiform cartilage, where presys- tolic murmur was heard; it was distinguished from first element of a double first sound, by absence of coincident impulse, by its preceding carotid pulse, and, especially, by its harsh quality.
9	Esie McK. ..	19	1	
10	Bridget G. ..	27	1	1	1	
11	John McG. ..	53	..	1	

* Not stated.

TABLE XII.—SUPPLEMENTARY.—*Concluded.*

No.	Name.	Age.	Hæmoptysis.	Dyspnoea.	Œdema.	Irregular and Inter- mittent Pulse.	Failure of Pulse.	Pneumonia.	Rheumatism.	Double 2nd Sound.	Accent. 2nd Sound. (in aorta)	Systolic Murmur at Apex.	Autopsy.	Other valves affected.	Form of Mitral Stenosis.	Complications and Observations.
12	Eliza B.	24	1	1	1	1	1	..	1	Sister to Kate B., No. 46, Table XI. Inadequacy of aortic valve, most probably by rup- ture of one or more of the segments. Partial left hemiplegia and left hemiplopia, by embolism; hemorrhage beneath left retina. Albuminuria and consecutive hypertrophy of the heart; endo-myocarditis, and systolic murmur. (See case 68.) Face occasionally livid, and swollen in the morn- ing. For last month has been cyanosed. Pulmonary emphysema. General hypertrophy of the heart; albuminuria and hæmaturia; no presystolic murmur during the thirty-nine days of patient's residence in hospital.
13	Thomas K.	24	1	1	1	1	1	..	1	Aortic inade- quacy.	..	
14	Mrs. B.	30	1	1	1	1	1	1	..	Mitral inade- quacy.	..	Face occasionally livid, and swollen in the morn- ing. For last month has been cyanosed. Pulmonary emphysema. General hypertrophy of the heart; albuminuria and hæmaturia; no presystolic murmur during the thirty-nine days of patient's residence in hospital.
15	Mary E.	56	1	1	1	..	1	Tricus- pid ste- nosis (?)	..	
16	Matthew N.	53	1	1	1	1	1	1	Mitral and Tri- cuspid Inade- quacy.	Button- hole.	Recurrent hæmoptysis; cyanosis; exophthalmia; syncope; presystolic murmur occasionally heard; and persistent postdiastolic murmur. Double impulse; increased area of precordial dullness; acute renal congestion; hæmaturia with albuminuria.
17	Mary E. C.	25	1	1	1	1	1	..	1	
18	Michael O'B.	21	1	1	1	1	1	1	..	1	..	Mitral inade- quacy.	..	
18			9	9	7	..	2	6	9*	4	5†	7	2			

* Not stated, 2.

† 3 in pulmonary artery, 3 in aorta.

It appears to me that some additional light may be thrown upon the statistics of mitral stenosis, and upon the relative frequency of its various complications, by a careful study and comparison of the results obtained from Dr. Hilton Fagge's cases, and my own as epitomised in Tables XI. and XII. For the calculations in both instances I am alone responsible. I have made them with the greatest care, and I hope they may be found at least substantially accurate.

I may here briefly state that Dr. Fagge's cases, 66 in number, are grouped under three heads, namely:

1. Those in which a presystolic murmur was heard during life, and the mitral orifice was found, after death, to be contracted; these cases were 7 in number.

2. Those in which the mitral orifice was found, on *post mortem* examination, to be contracted, but in which presystolic murmur had not been heard during life; the number of such cases was 40.

3. Cases in which a presystolic murmur was identified, but no opportunity was afforded for testing the diagnosis by *post mortem* examination; these cases numbered 19.

The fatal cases in this list were, therefore, 47 in number. A diagnosis was made in 7 instances, and verified by the result.

The total of my cases was 81. Out of this number 16 deaths took place, and the body was examined in 15 instances. The diagnosis of mitral narrowing was not made in 3 of the last-mentioned 15 cases, owing to the absence of presystolic murmur during the time the patients had been under observation. It was made in the 12 remaining cases, and in all it was verified by the result.

TABLE XIII.—ABSTRACT OF CASES OF MITRAL STENOSIS.

DR. FAGGE.		DR. HAYDEN.																																																																															
<p><i>Ages of Patients.</i></p> <table><tr><td>Under 10 years</td><td>...</td><td>0</td></tr><tr><td>10 and under 15 years</td><td>8</td><td>5</td></tr><tr><td>15</td><td>"</td><td>7</td></tr><tr><td>20</td><td>"</td><td>7</td></tr><tr><td>25</td><td>"</td><td>8</td></tr><tr><td>30</td><td>"</td><td>8</td></tr><tr><td>35</td><td>"</td><td>1</td></tr><tr><td>40</td><td>"</td><td>9</td></tr><tr><td>45</td><td>"</td><td>8</td></tr><tr><td>50</td><td>"</td><td>4</td></tr><tr><td>55</td><td>"</td><td>1</td></tr><tr><td>60 and over</td><td>...</td><td>2</td></tr><tr><td>Age not stated</td><td>...</td><td>5</td></tr></table> <p>— 66</p> <p>Total cases, 66.</p> <p>Average age of 61 patients = 32.77.</p>		Under 10 years	...	0	10 and under 15 years	8	5	15	"	7	20	"	7	25	"	8	30	"	8	35	"	1	40	"	9	45	"	8	50	"	4	55	"	1	60 and over	...	2	Age not stated	...	5	<p><i>Ages of Patients.</i></p> <table><tr><td>Under 10 years</td><td>...</td><td>2</td></tr><tr><td>10 and under 15 years</td><td>4</td><td>8</td></tr><tr><td>15</td><td>"</td><td>20</td></tr><tr><td>20</td><td>"</td><td>25</td></tr><tr><td>25</td><td>"</td><td>30</td></tr><tr><td>30</td><td>"</td><td>35</td></tr><tr><td>35</td><td>"</td><td>40</td></tr><tr><td>40</td><td>"</td><td>45</td></tr><tr><td>45</td><td>"</td><td>50</td></tr><tr><td>50</td><td>"</td><td>55</td></tr><tr><td>55</td><td>"</td><td>60</td></tr><tr><td>60 and over</td><td>...</td><td>0</td></tr><tr><td>Age not stated</td><td>...</td><td>4</td></tr></table> <p>— 81</p> <p>Total cases, 81.</p> <p>Average age of 77 patients = 29.63.</p>		Under 10 years	...	2	10 and under 15 years	4	8	15	"	20	20	"	25	25	"	30	30	"	35	35	"	40	40	"	45	45	"	50	50	"	55	55	"	60	60 and over	...	0	Age not stated	...	4
Under 10 years	...	0																																																																															
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60 and over	...	0																																																																															
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<p>Fetal cases in which the body was examined, 47.</p> <table><tr><td>Under 10 years</td><td>...</td><td>0</td></tr><tr><td>10 and under 15 years</td><td>2</td><td>1</td></tr><tr><td>15</td><td>"</td><td>20</td></tr><tr><td>20</td><td>"</td><td>25</td></tr><tr><td>25</td><td>"</td><td>30</td></tr><tr><td>30</td><td>"</td><td>35</td></tr><tr><td>35</td><td>"</td><td>40</td></tr><tr><td>40</td><td>"</td><td>45</td></tr><tr><td>45</td><td>"</td><td>50</td></tr><tr><td>50</td><td>"</td><td>55</td></tr><tr><td>55</td><td>"</td><td>60</td></tr><tr><td>60 and over</td><td>...</td><td>2</td></tr><tr><td>Age not stated</td><td>...</td><td>5</td></tr></table> <p>— 47</p> <p>Average age of 42 patients = 37.83.</p>		Under 10 years	...	0	10 and under 15 years	2	1	15	"	20	20	"	25	25	"	30	30	"	35	35	"	40	40	"	45	45	"	50	50	"	55	55	"	60	60 and over	...	2	Age not stated	...	5	<p>Fetal cases in which the body was examined, 15.</p> <table><tr><td>Under 10 years</td><td>...</td><td>1</td></tr><tr><td>10 and under 15 years</td><td>2</td><td>0</td></tr><tr><td>15</td><td>"</td><td>20</td></tr><tr><td>20</td><td>"</td><td>25</td></tr><tr><td>25</td><td>"</td><td>30</td></tr><tr><td>30</td><td>"</td><td>35</td></tr><tr><td>35</td><td>"</td><td>40</td></tr><tr><td>40</td><td>"</td><td>45</td></tr><tr><td>45</td><td>"</td><td>50</td></tr><tr><td>50</td><td>"</td><td>55</td></tr><tr><td>55</td><td>"</td><td>60</td></tr><tr><td>60 and over</td><td>...</td><td>0</td></tr><tr><td>Age not stated</td><td>...</td><td>1</td></tr></table> <p>— 15</p> <p>Average age of 15 patients = 29.26.</p>		Under 10 years	...	1	10 and under 15 years	2	0	15	"	20	20	"	25	25	"	30	30	"	35	35	"	40	40	"	45	45	"	50	50	"	55	55	"	60	60 and over	...	0	Age not stated	...	1
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<p><i>Sex of Patients.</i></p> <table><tr><td>Males</td><td>...</td><td>28 in 65 cases</td></tr><tr><td>Females</td><td>...</td><td>37 in 65 "</td></tr><tr><td>Sex not stated,</td><td>...</td><td>1</td></tr><tr><td></td><td></td><td>— 66</td></tr><tr><td></td><td></td><td>100.0</td></tr></table>		Males	...	28 in 65 cases	Females	...	37 in 65 "	Sex not stated,	...	1			— 66			100.0	<p><i>Sex of Patients.</i></p> <table><tr><td>Males</td><td>...</td><td>27 in 81 cases</td></tr><tr><td>Females</td><td>...</td><td>54 in 81 "</td></tr><tr><td></td><td></td><td>— 81</td></tr><tr><td></td><td></td><td>100.0</td></tr></table>		Males	...	27 in 81 cases	Females	...	54 in 81 "			— 81			100.0																																																			
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43.1 per cent. 56.9 "		33.3 per cent. 66.7 "																																																																															

TABLE XIII.—Continued.

DR. FAGGE.		DR. HAYDEN.	
<i>History of Symptoms.</i>		<i>History of Symptoms.</i>	
Antecedent Rheumatism ...	38 in 66 cases = 50.0 per cent.	Antecedent Rheumatism ...	40 in 81 cases = 49.3 per cent.
Hemoptysis ...	3 in 66 " = 4.5 "	Hemoptysis ...	44 in 81 " = 54.3 "
Irregularity of Pulse ...	16 in 66 " = 24.2 "	Irregularity of Pulse ...	15 in 81 " = 18.5 "
Reduplication of Second Sound ...	8 in 66 " = 4.5 "	Reduplication of Second Sound ...	30 in 81 " = 37.0 "
<i>Complications.</i>		<i>Complications.</i>	
Other valve lesions.	(Aortic obstruction ... 4)	Other valve lesions.	(Aortic obstruction, ... 2)
	Aortic obstruction and inadequacy ... 2		Aortic inadequacy ... 10
	Aortic obstruction & tricuspid inadequacy ... 1		Aortic obstruction and inadequacy ... 4
	Tricuspid inadequacy only ... 1		Tricuspid inadequacy ... 1
	Mitral inadequacy ... 14		Tricuspid obstruction ... 4
	Mitral inadequacy and aortic obstruction and inadequacy ... 2)		obstruction and inadequacy, with mitral inadequacy ... 1
			Mitral inadequacy ... 24
			Mitral and tricuspid inadequacy ... 1
			47 in 81 cases = 58.0 per cent.
Pulmonary apoplexy ...	6 in 47 cases = 12.7 per cent.	Pulmonary apoplexy ...	8 in 15 cases = 53.3 per cent.
Hemiplegia { Right, with aphasia ... 1 from embolism in total ... 2 Left, with aphasia ... 1 Slide not stated ... 1	5 in 66 cases = 7.5 per cent.	Hemiplegia { Right, with aphasia ... 5 from embolism in total ... 0 Left, with aphasia ... 0 Left, without aphasia ... 2	7 in 81 cases = 8.6 per cent.
Hemiplegia { from embolism in fatal cases.	4 in 47 cases = 8.5 per cent.	Hemiplegia { from embolism in fatal cases.	3 in 15 cases = 20.0 per cent.
Embolism of lower limb ...	3 in 66 cases = 4.5 per cent.		

In addition to those given in the preceding list, at least fifteen examples of mitral stenosis, accompanied by full details of *post mortem* examination, have been recorded in Ireland since 1855. In some of these cases a positive, and in others a presumptive, diagnosis was made.

The names of the authors, and the references to the record of the cases, arranged chronologically, are briefly as follows :

- DR. M'DOWEL : mitral stenosis ; no murmur audible ; diagnosis made from general symptoms. (*Proceedings of the Dublin Pathological Society.*) December 1st, 1855.
- DR. BANKS : aortic and mitral narrowing. (*Ibid.*) December 6th, 1856.
- DR. WILLIAM MOORE : mitral stenosis ; diagnosis of mitral disease. (*Ibid.* vol. i., part ii., p. 80.) March 24th, 1860.
- DR. LEES : mitral obstruction ; general diagnosis. (*Ibid.*, vol. i., part ii., p. 84.) April 14th, 1860.
- DR. STOKES : mitral, aortic, and tricuspid obstruction ; diagnosis of mitral narrowing. (*Ibid.*, vol. ii., part i., p. 14.) December 6th, 1862. (Case referred to at p. 1011.)
- DR. HEAD : mitral stenosis. (*Ibid.*, vol. ii., part iii., p. 165.) November 26th, 1864.
- DR. DUNCAN : mitral stenosis. (*Ibid.*, vol. ii., part iii., p. 186.) March 26th, 1865.
- DR. MACSWINEY : mitral stenosis. (*Ibid.*, vol. iii., part i., p. 132.) March 3rd, 1867.
- DR. HAWTREY BENSON : mitral and aortic obstruction, both diagnosed ; apex presystolic murmur identified. (*Ibid.*, vol. iv., part i., p. 35.) April 24th, 1869.
- DR. CRYAN : mitral and tricuspid obstruction ; diagnosis of double lesion ; presystolic murmur identified. (*Ibid.*, vol. iv., part ii., p. 144.) February 12th, 1870. (Case epitomised, p. 1013.)
- DR. MAGRATH (8th Hussars) : mitral stenosis. (*Ibid.* vol. iv., part iii., p. 250.) January, 1871.
- DR. CRYAN : mitral and tricuspid obstruction ; presumptive diagnosis of mitral stenosis from general symptoms ; no presystolic murmur audible. (*Ibid.*, vol. v., part i., p. 17.) April 20th, 1872. (Case epitomised, p. 899.)
- DR. GERALD YEO : extreme mitral constriction ; aortic obstruction and incompetency ; hypertrophy of the right ventricle ; hæmorrhagic infarction of the lungs, and extensive atheroma of the branches of the pulmonary artery in both lungs ; diagnosis of the twofold valve-lesion. (*Ibid.*, vol. iv., part ii., p. 123.) December 14th, 1872.
- DR. NIXON : mitral obstruction and aortic patency ; both diagnosed ; presystolic murmur recognized. (*Ibid.*, vol. v., part ii., p. 133.) February 8th, 1873. (See case 112.)
- DR. JOHN W. MARTIN, of Portlaw : mitral and aortic obstruction ; both diagnosed ; presystolic murmur recognized. (*Medical Press and Circular.*) March 11th, 1874.

I cannot bring my remarks on this subject to a conclusion without observing that, in 1827, Mr. Adams of Dublin antici-

pated most of what has been since done in regard to the diagnosis of mitral narrowing.*

That he recognised, at least in outline, the distinctive signs of this lesion, will appear from the following extract from his well-known memoir.† “When the ear is attentively applied to the side of the thorax, a very complex kind of movement, hard to describe, is heard. A hissing, purring noise, as it has been denominated, caused by the transmission of blood through a narrow orifice, is in most cases very evident.” He adverts to the dilatation and hypertrophy of the left auricle and right ventricle, and to the pulsation of the jugular veins, synchronous with that of the heart, and due to tricuspid reflux from inadequacy of the valves.

The fremitus and reduplicated second sound were described in the following words: “That the organ (heart) performed some kind of double movement, and that its motions were accompanied with a purring sound, as in varicose aneurism, was plainly audible, and also could be recognized when the hand was placed over the region of the heart.” His failure to appreciate the presystolic rhythm of the murmur and fremitus constitutes the only shortcoming in this graphic sketch. Thus, it would seem that, after Bertin, Dr. Adams was the first physician who identified mitral narrowing by its peculiar physical signs.

Inadequacy of the mitral valve from structural alteration of one or both of its segments, constitutes the most common form of valvular lesion.‡ This is due to the circumstance that the mitral valve is liable, not only to disease and injury proper to itself, but likewise to the consequences of retrograde pressure from disease at the orifice of the aorta.

The mitral valve may, however, be incompetent without having undergone any change of structure whatever. Cases belonging to the latter category may be reduced under the following heads: (a) Valvular inadequacy and regurgitation from displacement of one or both segments of the valve, through the papillary muscles and tendinous chords, by excentric yielding of the walls of the left ventricle at the acme of systole. This result

* This eminent man has just departed from amongst us (January, 1875).

† *Dublin Hospital Reports*, vol. iv.

‡ See p. 815.

may be due to fatty degeneration of the left ventricle, as exemplified in Case 49, p. 685; and in Case 50, p. 687. The remarks upon this subject at pages 282, 558, 562, and 690, may be referred to as explanatory of my view of the pathology of reflux from this cause. It may be likewise attributable to debility, without structural alteration of the valves or of the ventricle, from anæmia, malnutrition, or asthenic disease.* (b) Regurgitation from rupture of one or more of the primary tendinous chords.† (c) From dilatation of the left ventricle without proportionate elongation of the papillary muscles.‡ (d) Weakness and relaxation of the papillary muscles.§ (e) Adhesion of the tendinous chords to the walls of the ventricle.|| (f) Engagement in the orifice, of a pedunculated mass of fibrin, depending from its attachment to one of the curtains of the valve, or to the interior of the auricle.¶ (g) Fixation of the anterior segment of the mitral valve by a calcareous mass descending from the aortic orifice, and attached to the valve.** For the reasons already given, I cannot admit, as a cause of mitral reflux, spasm or irregular action of the papillary muscles;†† nor can I recognize murmur from excessive tension of the valves, without regurgitation.‡‡

In the great majority of cases, inadequacy of the mitral valve arises from antecedent inflammation and morbid alteration of its structure. Rheumatic endocarditis is the ordinary cause of the change just referred to. Inflammatory irritation consecutive to disease of the aortic valves, may lead to the same result. This is the cause to which active disorganization of the mitral valve,

* See pages 275, 282; Case 96, p. 881; also a case by Dr. Grimshaw, *Proceedings of the Pathological Society of Dublin*, vol. vi., part i., p. 18.

† See p. 235; Case 24, p. 535; also a case by Dr. Gordon, *Stokes on Diseases of the Heart*, Case 68, p. 473; and a case by Dr. A. W. Foot, *Proceedings of the Pathological Society of Dublin*, vol. vi., part i., p. 15.

‡ See p. 278-9.

§ See p. 280.

|| Niemeyer, *Opus citat.*, vol. i., p. 352.

¶ Walse, *Diseases of the Heart*, fourth edition, p. 93. An example of this kind was exhibited to the Pathological Society, by Dr. R. McDonnell, in Dec., 1871.

** Bristowe, *Transactions of the Pathological Society of London*, vol. iii., p. 157.

†† See p. 276.

‡‡ See p. 280.

of non-rheumatic origin, is in most cases to be attributed; the exceptions would include examples of uræmic, septicæmic, and traumatic valvulitis.

Inadequacy of the aortic valves, by the increased strain upon the walls of the left ventricle to which it gives rise, determines, at once, dilated hypertrophy of that chamber, and thickening by interstitial growth, of the mitral valves. In consequence of the structural changes which the valves have thus undergone, they are incapable of expanding proportionately to the further increase in the capacity of the ventricle, and mitral regurgitation is the necessary result, as Dr. Milner Fothergill states.* That in such cases, dilatation of the left ventricle is the principal factor of the mitral inadequacy, is shown by the absence of the latter lesion in cases of simple hypertrophy from renal disease, or from functional excitement.

Doctor Fothergill holds that in such cases the mitral valves are closed, and the strain upon them continued during ventricular diastole, by the reaction of the aorta. I am not satisfied upon this point. I apprehend that if such were the case, a diastolic murmur of mitral origin should be an invariable precursor of consecutive mitral inadequacy; but that it is not so related to the latter lesion is well known.

The pathology and symptoms of acute endocarditis, to whatever cause due, have been already discussed.† The mitral valve, rendered incompetent by inflammatory changes, is always thick and opaque. Whilst still affected by acute inflammation, the segments are swollen, but even on the surface, and of a uniform crimson hue; at a later stage they are of a dull white colour, more or less irregular on the surface, and firm in texture. The subsequent changes of the ordinary character consist in shrinking and retraction, with increased thickening and rigidity of the valve-segments.

The density of structure may attain to cartilaginous hardness, and in gouty subjects deposition of lime-salts and calcareous change may ultimately ensue. In this latter contingency, the surface and edges of the valves are rendered rough and spicu-

* *Lancet*, May 16th, 1874.

† See pp. 802-8.

lated, and so rigid, that obstruction usually coincides with inadequacy of the valves. The changes above mentioned, except calcification, may extend to the tendinous chords.

Inadequacy of the mitral valve necessarily involves systolic reflux upon the left auricle. The contractile force of the left ventricle is thus expended in part upon that chamber, which soon exhibits the effects of increased pressure in the consequent changes of dilatation and hypertrophy. During ventricular systole the left auricle is slowly filled, and within the measure of its normal capacity, by influx from the pulmonary veins only; but when the mitral valve is inadequate, a volume of blood, representing an undetermined but most probably a very small portion of the contents of the left ventricle, is superadded, and with a force and suddenness equivalent to the contractile energy of the left ventricle. The left auricle, stimulated by distention, reacts with increased energy, and propels the large volume of its contained blood with corresponding force upon the now dilating ventricle. The ventricle, being itself thus surcharged, contracts with vigour, and returns a portion of its contents upon the auricle. The two chambers thus alternately dilate and distend one another, and so become reciprocal stimulants to contraction.

The process thus continued must lead to dilatation and hypertrophy of both cavities, and within a period corresponding to (a) the degree of valvular inadequacy, and (b) the activity of nutrition. The right ventricle now begins to experience, through the pulmonary vessels, the effects of retrograde pressure from the left side; it undergoes dilatation, and, to a certain extent, hypertrophy, in a corresponding ratio. Flint remarks, that the coexistence of pulmonary emphysema will cause still greater enlargement of the right chambers than mitral inadequacy alone is capable of effecting.*

Hypertrophy is limited only by nutrition, which, to be adequate, must increase in a ratio proportionate to the growth of the walls. Ultimately, from advancing age, from an inadequate dietary, from excessive stimulation of the heart by labour, alcohol, or emotional excitement, or from defective supply of arterial blood to the walls of the heart, the balance of nutrition

* *Diseases of the Heart*, second edition, p. 142.

is deranged, and degeneration of tissue quickly follows. Dilatation now proceeds with increased rapidity; blood-stasis and engorgement, first of the pulmonary, and then of the general venous system, ensue; and the last stage, that of dropsy, is close at hand.

Niemeyer held that hypertrophy is consecutive to dilatation of the left ventricle, and that both these changes are of later occurrence than similar changes in the left auricle, because dependent upon them.* It is manifest, however, that there is, virtually, no pathological sequence whatever, but rather action and reaction, as between the two chambers. That the dropsical accumulations are precipitated by spanæmia from obstruction of the thoracic duct at the acme of venous engorgement, as urged by that author, I can readily admit. It is through this channel that the fat, the fibrin, the nascent corpuscles, and in part the albumen of the blood are introduced into the circulation.

As a consequence of dilatation of the left auricle, the pulmonary veins are enlarged, and their apertures of entrance into the auricle, much dilated; by retrograde pressure, the branches and trunk of the pulmonary artery undergo a similar change. Dr. Milner Fothergill, adopting the opinion of Virchow, believes that atheroma of the pulmonary artery and its branches, is amongst the usual remote consequences of mitral regurgitation, and is due, in his opinion, to interference with the circulation in the bronchial and pulmonary vessels from excessive vascular tension.† Dr. Gerald Yeo has exhibited before the Pathological Society of Dublin an example of extensive atheroma of the pulmonary artery; it was taken from the body of a woman who had died from the consequences of extreme mitral narrowing: "The pulmonary artery, from the semilunar valves down to the very finest subdivision, was irregularly dilated and inelastic; the inner coat throughout being studded with hard, prominent, yellow patches, some of which were rough on the surface, and looked like ordinary atheromatous ulcers."‡

* *Text-Book of Practical Medicine*, by Humphreys and Hackley, 1869, vol. i., p. 355.

† *The Heart and its Diseases*, 1872, p. 129.

‡ *Proceedings of the Pathological Society of Dublin*, new series, vol. v., part ii. p. 125.

Tension of the pulmonary vessels, though primarily dependent upon reflux from the left side of the heart, is aggravated by the *vis a tergo* from the right ventricle. The irritation to which this latter chamber is subjected from continued engorgement and tension, soon leads to dilatation of its cavity, and thickening, usually but slight, of its walls. On the other hand, these changes in the right ventricle lead by reaction to increased vascular tension in the lungs. Thus, as between the left auricle and ventricle, action and reaction, followed by the usual consequences of mutual stimulation, are exhibited in the relationship of the pulmonary vessels to the right ventricle, in cases of mitral inadequacy. Distention of the right ventricle implies a similar state of the right auricle, parietal congestion of the heart, and engorgement of the trunks and tributaries of the cavæ. The ulterior consequences of these changes are soon exhibited in general venous congestion, enlargement of the liver, stasis of the portal system attended with anorexia, nausea, and foul tongue, and frequently with enlargement of the hæmorrhoidal veins; congestion of the kidneys, with a diminished secretion of concentrated and high-coloured urine loaded with lithates; and finally, anasarca and effusion into the great serous cavities. At a comparatively early stage the tricuspid valves become inadequate by dilatation of the right auriculo-ventricular orifice, and pulsation of the jugular veins is exhibited. This is of two kinds, namely, a strong pulsation synchronous with ventricular systole; and a series of feeble undulations, terminating in a wave of greater magnitude, corresponding to the contractions of the right auricle.*

Niemeyer, endorsing the statement of Bamberger, maintains that even before tricuspid inadequacy and distention of the jugular veins has taken place, rhythmical undulations of these vessels may be often witnessed as a consequence of transmitted vibration from the tricuspid valve.†

The pulse of mitral regurgitation is in no respect characteristic. Hope maintained that when mitral inadequacy of even slight degree exists, the pulse is always somewhat weak and intermit-

* See pp. 500 and 571.

† *Opus citat.*, p. 355.

tent.* Herein he was undoubtedly in error. The pulse is always feeble in proportion to the degree of mitral reflux, but irregularity and intermittence are late characteristics of it, and may be regarded as evidence of failure of the heart. Walshe agrees with Marey in the opinion that coincident mitral stenosis may render the pulse of mitral regurgitation regular, and he further suggests that aortic obstruction may have the same effect. But the pulse, even in the absence of these complications, is usually regular in the early stages of the disease; and in the later stages, it is quite as frequently irregular where they coexist. In fine, I look upon irregularity and intermittence of the pulse, when present in the disease under consideration, as symptoms, not of the valvular inadequacy, but of one of its latest and most formidable sequelæ, debility and failure of the left ventricle.

There are no symptoms suggestive of mitral insufficiency in its early stages, with the exception of palpitation and breathlessness on exertion, and these may be due to so many other causes, pulmonary, hæmic, and nervous, that they must be regarded as possessing very little if any positive value. In the absence of any of these causes, and associated presumptively with a history of antecedent rheumatic affection of the heart, their value will be somewhat enhanced, as pointing to mitral inadequacy.

Congestion of the lungs is amongst the earliest of the advanced symptoms of this affection; it is indicated by dyspnoea and short teasing cough, accompanied by thin serous expectoration; occasionally, there is a streak of blood in the sputum, and, still more rarely, it consists entirely of blood. Hæmoptysis is, according to my experience, by far more common in connexion with mitral narrowing than with simple mitral inadequacy.

All the symptoms, then, which are usually associated with the ulterior stages of mitral regurgitation are no less suggestive of mitral obstruction, and a few of them point even more directly to the latter affection. Hence, the differential diagnosis must rest upon physical signs exclusively. These are, however, sufficiently distinctive, and may be classified under the two heads of signs belonging to the early, and signs appertaining to the later

* *Diseases of the Heart*, p. 376.

stages of the disease. Murmur and thrill constitute the former group; and these, supplemented by the signs of hypertrophy, dilatation, engorgement, and failure of the heart, make up the latter signs.

Murmur is the most distinctive, and, taken alone, the only pathognomonic sign of mitral regurgitation. It is usually soft and blowing in quality throughout; but occasionally, even in the nascent stage of its development, it is associated with a musical note which is more or less faint, and always, when not substitutive, is terminal in relation to the murmur. At first, and for a more or less protracted period, according to the rate of progress of ulterior changes in the valves and in the heart, the murmur accompanies the first sound, and is, in many cases, at this period postsystolic rather than systolic in rhythm. At a later date, and coincidently with the evidence of utter disorganization of the valves and failure of the left ventricle, it replaces the first sound and becomes substitutive.

In regard to diffusion and transmission, mitral systolic murmur in the adult is strictly limited to the mitral area previous to the development of consecutive hypertrophy, when extrinsic causes of diffusion, such as a solidified lung, do not exist; but in the child, owing to the high conducting properties of the thoracic parietes, it is diffused far beyond these limits, even at the earliest period. In those rare cases in which the valvular lesion is consecutive to hypertrophy from chronic renal disease, atheroma of the aorta, or functional excitement, and where it follows disorganization of the aortic valves, the murmur is quite as extensively diffused from the beginning as it is in the advanced stages of primary mitral lesion.

It has been held by eminent authority that the murmur of mitral regurgitation "of structural mechanism" is almost necessarily audible at the angle of the left scapula and in the left interscapular space, and necessarily so in cases of "well-pronounced organic regurgitation."* I can subscribe the former proposition only in the limited sense above indicated; but the latter I accept in its entirety. Dr. Andrew says: "If the murmur be not audible in this region (at or near the inferior angle

* *Walsh, Diseases of the Heart and Great Vessels*, fourth edition, 1873, p. 94.

of the left scapula), I believe that it rarely indicates regurgitation.* Murmur was heard in this situation in 66 of the 100 cases of mitral regurgitation reported by him; but in 64 of these he admits that hypertrophy likewise existed.

Of the 25 cases given in Table XIV., p. 987, most of which were noted many years ago, and before I had been impressed with the value of left scapular murmur as a sign of mitral regurgitation, no record of this sign was made, I regret to say, in 12 instances. It was present in 9 of the cases, all of which likewise exhibited indubitable evidence of hypertrophy, whilst it was absent in 4; and of these, all, with one exception (No. 22), were recent cases.† In the exceptional instance just referred to, there was likewise mitral obstruction, and *hypertrophy did not exist.*‡

I therefore hold, that whilst the existence of a systolic murmur at the angle of the left scapula, coinciding with one of the same rhythm at the apex of the heart, must be regarded as affording confirmatory evidence of mitral regurgitation with hypertrophy of the left ventricle, the absence of it is quite consistent with mitral inadequacy of organic mechanism before hypertrophy has supervened.

Accentuation of the second sound in the pulmonary artery has been held to be an all but essential sign of mitral regurgitation. Skoda so regarded it, and in the absence of this sign, he would have sought an explanation of systolic mitral murmur in assumed roughness of the mitral valve on its ventricular surface, or of the endocardium in the vicinity of the aortic orifice.§

He adds, "An increase in the second sound of the pulmonary artery almost invariably accompanies defect of the mitral valve. I believe that it can only fail to do so when the coats of the pulmonary artery have lost their elasticity, and do not contract rapidly after distention."||

Doctor Walshe does not regard this sign as necessarily present in mitral regurgitation. He declares that he has often known it

* *St. Bartholomew's Hospital Reports*, vol. i., p. 15.

† Nos. 18, 19, and 21.

‡ See Case 102; also p. 232.

§ *On Auscultation*, by Markham, p. 229.

|| *Opus citat.*, p. 230.

to be wanting where mitral inadequacy existed, and tricuspid regurgitation could not have been adduced to explain its absence on the assumption of diminished pressure.* The pulmonary second sound was intensified in no less than 70 out of the 100 cases reported by Dr. Andrew.† Nevertheless, as he judiciously remarks, the intensification may be only relative, the second sound in the aorta being diminished in vigour by reflux at the mitral orifice; or it may be apparent, either from masking of the aortic sound, or from favourable conduction of that of the pulmonic artery. Finally, inasmuch as hypertrophy of the right ventricle is admitted by Skoda to be the condition upon which intensified pulmonary second sound essentially depends, and this may be due to causes other than disease at the mitral orifice, *e.g.*, pulmonary emphysema, chronic bronchitis, or pressure upon the trunk of the pulmonary artery, an accentuated pulmonary second sound may likewise depend upon any of these causes. It should be added, that mitral reflux must fail to produce an intensified sound in the pulmonary artery previously to the development of right ventricular hypertrophy; and that it may fail to have that effect even when associated with hypertrophy, owing to impaired elasticity in the coats of the pulmonary artery, as Skoda remarked. On reference to Table XIV., it will be seen that the phenomenon under consideration existed in 7 only of the 25 cases included.

Fremitus or thrill has been regarded by Hope as a frequent attendant of mitral regurgitation.‡ The declaration, however, with which he accompanies this statement, namely, that thrill is less often associated with obstruction than with inadequacy at the mitral orifice, must raise a doubt as to the accuracy of the diagnosis upon which it was based.

Bertin§ declares that tactile fremitus and hoarse murmur (*bruissement*) are "infallible signs" of contraction of the orifices of the heart. This assertion is too general, as both these signs may, although they rarely do, coincide with regurgitation. Tactile fremitus is of frequent occurrence in connexion with mitral stenosis,|| whilst, of the 25 cases included in Table XIV.,

* *Opus citat.*, p. 94.

‡ *Opus citat.*, p. 387.

† *Loco citat.*, p. 16.

§ *Traité des Maladies du Cœur*, p. 199.

|| See Tables XI. and XII., pp. 964, 967.

it was present in 2 only. It is, in truth, of exceptional occurrence in connexion with simple mitral inadequacy, as might have been presumed from the character of the lesion to which defect of the mitral valve is ordinarily due. When present, it depends upon a rugged and rigid state of the valves, or upon a flake of fibrin entangled in the orifice, and vibrating in the re-fluent stream. In the former of these contingencies the associated murmur would be harsh, and in the latter it would be soft in quality.*

The evidence upon which the diagnosis of hypertrophy and dilatation of the ventricles should rest has been already pointed out;† the signs of enlargement of the left auricle, however, demand a special notice here. Owing to the deep-seated position of this chamber, the tactile phenomena, indicative of increase in its volume or in the force of its contraction, are of necessity but imperfectly pronounced, and liable to be misinterpreted. A strong pulsation with increased dulness in the second or third intercostal space, immediately inside the left nipple-line, has been regarded as distinctive of dilated hypertrophy of the left auricle; if this pulsation coincide with the presystole, it is held to indicate enlargement of the auricle due to mitral obstruction; if it be systolic in rhythm, it is regarded as diagnostic of a similar condition of the auricle from mitral reflux. I must confess that I am not satisfied as to the alleged value of these signs. The coincident enlargement of the ventricles, or of either of them, must render diagnosis based upon the special localization of the left auricle, very uncertain; and, indeed, the doubtful aid to diagnosis which the phenomena in question are capable of affording in ordinary examples of either lesion can be well dispensed with. In extreme cases they may be so well pronounced as to leave no room to doubt their significance. A case in which they were of this character has been published by Dr. Stokes. A girl, aged eleven years, who had had a well pronounced mitral regurgitant murmur, and was subject to attacks of cardiac asthma, was visited by Dr. Stokes in one of these attacks, which was of unusual severity, and attended with extreme rapidity of pulse and of respiration; the left side of the chest was absolutely dull, ex-

* *Vide* Nos. 15 and 28 in Table XIV.

† *See* p. 493, and p. 561.

cept in its posterior and inferior portion, whilst the action of the heart was in the highest degree irregular. The positive and negative evidence led Dr. Stokes to conclude that the dulness of the side was due to temporary but extreme distention and enlargement of the left auricle.*

Mitral regurgitation, accompanied by murmur, although most frequently due to thickening or shortening of the valves, or vegetations upon their edges,† has been likewise attributed to the several causes mentioned at p. 973. I have not met with examples of mitral reflux murmur, arising from the causes mentioned by Niemeyer, Walshe, and Bristowe.‡ I have already shown (p. 289) that it is scarcely possible to mistake for a murmur of mitral regurgitation, one due to roughening of the ventricular surface of the mitral valve, or of the surface of the ventricle near the orifice of the aorta. The murmur in such a case, although audible at the apex, would be of maximum intensity at the base, and audible by transmission in the ascending aorta and its primary branches.§ A systolic murmur of a musical character, occupying the area and the line of transmission just indicated, has been attributed by Dr. R. St. John Mayne to the vibration of an irregular tendinous chord stretched across the mouth of the aorta, and attached by both its extremities to the walls of the ventricle. In this case, however, the aortic orifice was obstructed by calcareous change of the sigmoid valves.||

Cysts may be formed upon the mitral valve, and give rise to a murmur of inadequacy. An example of this kind, which proved rapidly fatal by rupture of the cyst and septicæmia, has been reported by Dr. Day of Dulwich.¶

Inadequacy of the mitral valve suddenly produced by rupture of one of its cusps, or of the attached tendons or papillary muscles, can scarcely occur where antecedent degenerative changes of the structures involved have not been in operation. The symptoms are those of acute congestion of the lungs by urgent

* *Diseases of the Heart and Aorta*, p. 204.

† See p. 233.

‡ See p. 974. Various other alleged causes of mitral reflux murmur have been mentioned by authors. See pp. 243, 275, and 280.

§ See Case 31, p. 553.

|| *Proceedings of the Pathological Society of Dublin*, vol. iv., part i., p. 27.

¶ *Biennial Retrospect of Medicine and Surgery*, 1867.

dyspnoea, oppression, and hæmoptysis, accompanied by weakness and irregularity of the pulse, and remotely followed by anasarca. The pulmonary symptoms, as justly remarked by Dr. Todd,* are more urgent in connexion with rupture of the mitral valve or its sustaining appendages, than with a corresponding accident at the tricuspid orifice. A loud systolic bellows-murmur of mitral origin, and accompanied or not by a musical note, constitutes the principal indication of this accident. Murmur of this rhythm and site, not previously existing, and coinciding in development with the symptoms above mentioned, would warrant a positive diagnosis of rupture of the mitral valve or its appurtenances.

The treatment of mitral inadequacy should be directed mainly to the consecutive changes of dilatation of the left ventricle and debility of its parietes. The object of treatment should be to avert the latter contingency, upon which failure of the arterial circulation, pulmonary and general venous congestion, and dropsy, directly depend. Whilst moderate and gentle exercise and a generous but non-stimulating diet should be prescribed with a view to the promotion of nutrition, great muscular exertion, especially such as involves strain and temporary suspension of breathing, and the free use of alcohol, must be forbidden. Indulgence in the use of tobacco, tea, and coffee, as eminently enervating, should be kept within moderate limits. Straining at stool must be avoided; and hence the necessity for occasional aperients, and the use of vegetable food and other alimentary substances which tend to avert constipation. Moderate use of good wines of the tonic class, as claret, etc., may be allowed.

Of medicinal agents, iron, digitalis, quinine, and strychnia are the most valuable; the two former may be given in combination, alternately with the two latter. Thus, ℥v-x of the tincture of the perchloride, or of the acetate, of iron, may be administered thrice daily in an ounce of the infusion of quassia or calumba; or grs. ij of quinine with ℥v of the pharmacopœial solution of strychnia may be given in solution, with a few drops of dilute sulphuric acid, at corresponding periods. For the preceding, a teaspoonful of the syrup of the triple phosphates (iron, quinine, and strychnia) may be substituted. The treatment of pulmonary and general congestion, and dropsy, should be con-

* *Dublin Journal of Medical Science*, new series, vol. v.

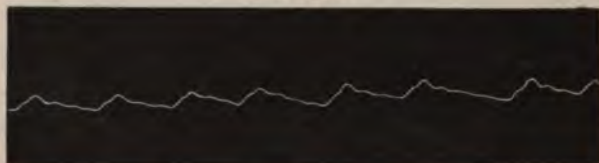
ducted on the ordinary principles. As a diuretic, digitalis, in combination with spirit of nitrous ether, is the best; but as preliminary measures, dry cupping and warm poulticing to the loins, and free purgation, should be resorted to, with a view to the disengorgement of the kidneys.

Hæmoptysis is by no means an ordinary symptom of mitral inadequacy prior to the stage of passive congestion of the lungs; and even then, it is rarely excessive. In this respect, mitral obstruction presents a striking contrast. A streak of blood in the sputa is not, however, an unusual accompaniment of the intercurrent bronchitis of mitral inadequacy.

Of the 25 cases summarized in Table XIV., p. 988, expectoration of blood, even in the smallest quantity, occurred in 7 only. When not excessive, hæmoptysis requires no special treatment; attention should be given mainly to the condition of the lungs upon which it depends. When a styptic is demanded, the liquid extract of ergot, in ℥xv doses, or ergotin used hypodermically, is the most efficacious.

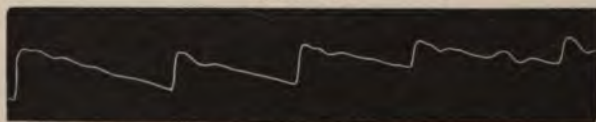
The annexed tracings (Figs. LXV. and LXVI.) exhibit the graphic character of the pulse of mitral regurgitation. Fig. LXV. shows the state of imperfect arterial repletion and defective tension, with regularity of rhythm, which may be witnessed in the early stages of the disease, whilst Fig. LXVI. represents the hypertrophy, with incipient failure of the left ventricle from tissue-degeneration, which characterizes its more advanced stage. (see p. 978.)

FIG. LXV.



Mitral Regurgitation (early stage). Mary M.

FIG. LXVI.



Mitral Regurgitation (late stage). No identification.

TABLE XIV.—MITRAL REGURGITATION.

No.	Name.	Age.	Occupation.	Hemiplegia.	Dyspnea.	Edema.	Rheumatism.	Irregular or Falling Pulse.	Frenitus.	Inten. 2nd Sound in Pulmonary Artery.	Reduplicated 2nd Sound.	Pitch, Quality, and Rhythm of Murmur.	Murmur Audible in Left Back.	Result.
1	Pat. M. ..	24	Turner in wood ..	1	1	1	1	1	Rough and musical, substitutive systolic	*	Discharged.
2	Thomas B. ..	14	Sailor-boy ..	1	1	1	..	Soft and accompanying, post-systolic	*	Do.
3	Eliza O'B. ..	54	Dressmaker	1	1	..	1	..	Systolic, substitutive, and musical	*	Do.
4	Eliza McG. ..	54	1	1	1	..	Soft, systolic and accompanying	*	Do.
5	Jane E. ..	15	None	1	Systolic, rough, and accompanying	*	Do.
6	Jane R. ..	22	Domestic servant ..	1	1	Soft, systolic, and accompanying	*	Do.
7	John McG. ..	22	Merchant's clerk	1	Soft, systolic, and accompanying	*	Do.
8	Maryanne C. ..	17	None	1	1	1	1	Soft, systolic, and accompanying	*	Do.
9	Luke McC. ..	63	Butler	1	1	1	1	Rough and accompanying, post-systolic	1	Death (see Case 121).
10	Susanna F. ..	26	None	1	1	1	Systolic, soft substitutive	1	Discharged.
11	William G. ..	15	Shoemaker	1	1	..	1	..	Substitutive systolic & <i>de cade</i>	1	Death (no p. m.).
12	John M. ..	21	Van-driver	1	1	..	1	..	Soft, systolic, and substitutive	1	Discharged.
13	Lawrence O'B. ..	24	Paper manufacturer	1	1	1	1	Soft, substitutive, systolic	*	Do. (pericarditis).
14	Christopher M. ..	54	Labourer	1	1	Soft, systolic, and accompanying, with musical note	1	Do.
15	Catherine O'N. ..	13	None	1	Scar-latina	..	1	Rough, systolic accompanying	1	Do.
16	George R. ..	13	None	1	1	1	Soft, substitutive systolic	*	Do.
17	Maryanne C. ..	19	Domestic servant ..	1	1	1	1	Soft, systolic, and accompanying	*	Death, hypertrophy, and renal disease (see Case 27).
18	Eliza D. ..	15	None	1	Musical systolic	..	Discharged. Disease recurrent.
19	Mary M. ..	60	Domestic servant	1	Soft, substitutive systolic	*	Discharged.
20	Magaret C. ..	46	Charwoman ..	1	1	1	1	Soft, substitutive systolic	..	Do.
21	Anne G. ..	60	Children's maid	Harsh, substitutive systolic	..	Do.
22	Anne K. ..	30	None ..	1	1	1	1	1	1	1	..	Soft, systolic, and accompanying	..	Do.
23	Thomas O'N. ..	15	None	1	1	1	1	Soft, substitutive systolic	1	Do.
24	Catherine M. ..	42	None	1	1	1	1	..	1	..	Soft, substitutive systolic	1	Do.
25	Martin M. ..	42	Porter ..	1	1	1	1	1	..	1	..	Soft, substitutive systolic, ending in a musical note	1	Do.
25	11 males, 14 females.			7	11	14	16	7	2	7	0		9	Died 3 Discharged 22.

CASE CXVIII.—*Acute Rheumatism with Hæmoptysis; Precordial Pain; Palpitation and Rough Presystolic Murmur at Apex; Accentuated Second Sound. Diagnosis: Acute Endocarditis with Mitral Regurgitation.*

Thomas B., a sailor-boy, aged fourteen years, was admitted May 23rd, 1865. A month previously he caught cold, which was followed by articular pains, also by pain in the left side and palpitation on exertion. On the day preceding that of admittance, he had spat a good deal of florid blood. When admitted, his condition was as follows: Pulse 96, weak, but regular; heart acted tumultuously; a rough murmur, confined to the area of the apex, succeeded the first sound. The second sound was normal. To have grs. ij of mercury with chalk, thrice daily.

28th. An apex-murmur, of the character and rhythm just described, occupied the entire short pause.

June 1st. Pulse steady, and 102 in erect posture. A sharp grating murmur, strictly confined to the area of the apex, succeeded the first, and ran quite up to the second sound, which was accentuated.

5th. Gums slightly affected by the mercury, which was stopped. An astringent gargle was directed, also grs. v of iodide of potassium, thrice daily.

The boy was discharged towards the end of June, free from urgent symptoms, the apex-murmur, however, retaining its previous characters.

CASE CXIX.—*History of Rheumatism; Palpitation; Great Weakness; Irregularity of Pulse; Displacement of the Apex of the Heart Outwards; Systolic Apex-murmur, which was occasionally attended with a Musical Note; Second Sound Accentuated in the Pulmonary Artery; Repeated Admittances, followed by Temporary Improvement. Diagnosis: Mitral Inadequacy; Hypertrophy, with Dilatation and Tissue-degeneration of Left Ventricle.*

Eliza O'B., a dress-maker, aged fifty-four years, admitted May 15th, 1865. Has suffered occasionally from rheumatic pain in the hips, and about four years ago began to suffer from palpita-

tion and weakness; has never spat blood; is remarkably weak and desponding. Pulse 84, weak, and very irregular. The apex of the heart pulsates an inch and a-half to the outside of, and below the nipple-line; a loud bellows-murmur is heard to accompany the first sound at the apex, and occasionally the latter portion of this murmur is attended with a musical note. The second sound is accentuated at the junction of the third costal cartilage with the sternum on the left side. A bitter tonic with iron was prescribed, with generous diet and stimulants.

She was discharged on the 10th of June, her general condition being then much improved, but the cardiac signs unaltered.

30th. Visited hospital as an extern; was much agitated after a "fright."

She was re-admitted in the early part of May, 1866, being then in a state of great debility; there was slight bronchitis. Action of heart irregular and intermittent; systolic murmur at left apex.

Discharged, at her own request, May 16th. She then felt much better, but the action of the heart was still irregular, and a murmur, as previously described, was audible at the apex.

29th. Admitted again in a state of extreme debility. Pulse 90, weak, and remarkably irregular. The action of the heart was likewise irregular, and occasionally its contractions were not represented in the radial artery.

She was discharged on the 10th of June. There was then no dyspnoea or oedema, but the physical signs were as last described.

This patient was admitted for the last time on the 22nd of June, in a state of extreme weakness, and discharged on the 28th, in a somewhat improved condition.

CASE CXX.—*Displacement of Apex of Heart Outwards; Precordial Pain and Distress; Extension of Precordial Dulness; Rough Systolic Murmur, with Suppression of Second Sound at Apex; Accentuated Second Sound at Base. Diagnosis: Hypertrophy of Left Ventricle; Roughening and Inadequacy of Mitral Valve.*

Jane E., aged fifteen years, admitted June 23rd, 1865. Never

had rheumatism, and never spat blood. Eight months previously she began to suffer from palpitation, and six months later from cough. Pulse 90, regular. Precordial dulness four and a-half inches vertically, by four inches transversely. Apex-beat removed somewhat to left. Urgent precordial pain; cardiac impulse strong; no fremitus; a loud jarring murmur is heard with the first sound at the apex, where no second sound was audible. At the base, both sounds were heard without murmur; the first ill pronounced, the second accentuated. One leech to be applied over heart. To have ℥v of chloric ether in half an ounce of camphor-water every fourth hour.

30th. Is free from cardiac pain and distress, and can lie on left side; murmur less rough.

This patient passed under the care of my colleague, Dr. Hughes, on the 1st of July.

She was re-admitted on the 1st of November following. The feet were then swollen; she had cough, and was in a state of urgent distress from cardiac asthma. Pulse 132, regular, but small. Physical signs as before. Leeches were applied over the heart; and iron, with tincture of digitalis, was given.

CASE CXXI.—Rheumatic Gout; Tortuosity and Visible Pulsation of the Radial Arteries; Intermittence of the Heart; Rough Postsystolic Murmur at Apex, and likewise audible in Left Back; Second Sound Suppressed at Apex, but Accentuated in the Pulmonary Artery; Re-admittance Seven Years later; Œdema of Feet and Legs; Great Debility; Death. Autopsy: Effusion into all the Serous Cavities; Congestion of Lungs and Condensation of the Right with Investment by False Membrane; Enlargement of the Heart; Hypertrophy of both Ventricles; Thrombosis of the Right Chambers and of the Pulmonary Artery; Inadequacy by Thickening of the Mitral Valve; Atheroma of the Aorta.

Luke McC., aged sixty-three years, an officer's servant, and had served in many climates, was received into hospital February 17th, 1866. Had been subject to rheumatic gout for the last two years.

When admitted, he was suffering from slight pain and swelling in ball of left great toe, and joints of right hand. The tongue was clean; the pulse 72, and slightly visible in the radial arteries, which were rigid and tortuous. Precordial dulness was not extended, and there was no fremitus. The heart acted regularly, and in the normal position. A loud systolic bellows-murmur was heard at the apex; it was extensively transmitted over the left side, was feebly audible in the back, and less distinctly so in the course of the aorta. No second sound was anywhere heard. To have ℥x of tincture of colchicum in an ounce of Murray's fluid magnesia, every third hour.

23rd. The heart occasionally intermitted; urine clear, acid, 1.020 in sp. gr., and free from albumen; skin moist; had then no pain in feet. The murmur at the apex was remarkably rough, and was *preceded* by the impulse of the heart. No second sound was audible at the apex, but it existed at the base, and was accentuated in the pulmonary artery.

26th. The murmur at the apex was distinctly postsystolic, immediately succeeding the impulse and the valve-click of the first sound. There was an occasional intermission, and also from time to time, an irregular and abortive impulse of the heart. Pulse 78. The man was then quite free from pain, and was discharged.

After an absence of more than seven years he was re-admitted in the early part of October, 1873, in a state of great debility. The feet and legs were then swollen. The breathing was occasionally paroxysmal. Urine normal. The heart acted irregularly; the impulse was weak, and the apex-pulsation was felt in the nipple-line; the sounds were faint, but *free from murmur*.

He died on the 14th of October. For several days preceding death, the radial pulse had been scarcely perceptible.

All the serous cavities were found full of serum. Both lungs were congested, and the right was condensed, and covered with old false membrane. The heart weighed twelve ounces, and the right chambers contained some blood-clot. From the right auricle a long cylinder of white fibrin passed into the right ventricle, and thence into the pulmonary artery.

Both ventricles were in a state of hypertrophy, the right being

half an inch, and the left three-fourths of an inch thick. The valves were healthy, with the exception of the anterior and right segment of the mitral, which was thick on the edge. The aorta exhibited patches of atheroma at the root, in the sinuses, and in the transverse portion of the arch.

CASE CXXII.—*Rheumatism ; Chronic Cough, with Hæmoptysis ; Dyspnoea ; Partial Syncope ; Oedema ; Irregularity of Pulse and of Respiration ; Visible Pulsation of Superficial Arteries, and Venous Pulsation in Neck ; Hepatic and Pulmonary Congestion, and Right Pleural Effusion ; Extension of Precordial Dulness, and, Nineteen Months later, Abolition of it from Pulmonary Emphysema ; a Substitutive Systolic Bellows-murmur Terminated by a Musical Note at Apex, likewise Audible in Left Back, all over Left Side, and in the Course of the Ascending Aorta ; Second Sound Accentuated in the Pulmonary Artery ; Three several Admissions, at Intervals of Six and Twelve Months, followed by Temporary Improvement. Diagnosis : Mitral Inadequacy ; Hypertrophy, with Dilatation of both Ventricles ; Fatty Degeneration of Substance of Heart ; Probable Atheroma of the Aorta.*

Martin M., aged forty-two years, a porter in the city fish market, was admitted into hospital on the 26th of April, 1872. Had rheumatic inflammation of the joints two years ago ; for the last three months has been subject to cough, dyspnoea on exertion, and occasionally to faintness, which has obliged him to sit down. The feet began to swell a week before admittance, up to which date he had continued at his work.

On the 27th he was very weak ; respiration was quick, shallow, and irregular ; the pulse was arhythmically irregular, and very feeble ; the pulsation of the radial and carotid arteries was slightly visible ; and there was venous pulsation synchronous with ventricular systole in the supraclavicular and suprasternal fossæ. The feet, legs, and genitals were swollen. The liver was enlarged and tender, and both lungs were congested posteriorly. Precordial dulness extended transversely from the nipple-line to the right margin of the sternum ; the cardiac impulse was dif-

fused and very feeble. At the point of apex-pulsation, which was in the nipple-line, a soft substitutive systolic murmur terminated by a musical note, was heard. At the right base both cardiac sounds were dull, but free from murmur; and at the left base the second sound was intensified. To have, thrice daily, tincture of digitalis, tincture of the perchloride of iron, and spirit of nitrous ether, of each ℥x, in an ounce of infusion of quassia. After a few weeks in hospital the man was free from dyspnoea and faintness; cedema had entirely disappeared, and he returned to his employment.

Re-admitted on the 8th of November, 1872. The lower limbs were swollen; the pulse very irregular; apex-pulsation was felt in the nipple-line. A loud, blowing, systolic murmur was heard at the apex, and at the base, a faint, transmitted murmur of the same rhythm; there was venous pulsation in the neck. After a short course of treatment, similar to that previously mentioned, he was relieved from his urgent symptoms, and was again discharged at his own request, in order to resume his work as a porter.

Admitted, for the third time, on the 2nd of December, 1873. The symptoms then were as follows: Cough and expectoration of mucus, which was occasionally streaked with blood; respiration 36 and shallow; pulse 72, failing, and irregular; partial effusion into right pleura, and cedema of left lung; downward displacement of liver; urine about sixteen ounces in the twenty-four hours. Physical signs: Precordium resonant, and cardiac impulse felt at xyphoid cartilage; a loud, musical, systolic murmur over the left side of the chest generally, in the left back, and likewise in the course of the ascending aorta; it was not transmitted into the arteries of the neck. The point of maximum intensity of this murmur was in the fifth intercostal space, half an inch inside the nipple-line. The carotids pulsated visibly, but there was no venous pulse in the neck.

He was discharged on the 20th of January, 1874, improved as to general symptoms, the physical signs, however, being as last described.

CASE CXXIII.—*Repeated Attacks of Rheumatism ; Strong Pulsation of Heart, and Loud Systolic Murmur at Apex and in Left Back ; Five Years later, Sudden Occurrence of Right Hemiplegia and Amnesic Aphasia ; Pulse Regular, but Dicrotus ; Ecthymatous Eruption on Face and Neck ; Partial Recovery of Motion in Paralyzed Limbs ; Syncope ; Death. No Autopsy. Diagnosis : Mitral Inadequacy of Rheumatic Origin, and subsequent Embolism.*

William G., aged fifteen years, a shoemaker's apprentice, was received into hospital, November 14th, 1861. He had had several attacks of acute rheumatism in childhood ; was pale and haggard, and not grown in proportion to his years ; heart's action strong, and visible pulsation in usual situation of apex. A loud systolic murmur was audible at the apex, and likewise in the left back ; the second sound was normal. He was discharged on the 28th December, being then able to resume his work.

In the course of the succeeding five years, this boy repeatedly presented himself at the hospital as an extern patient, and I had many opportunities of examining him. Within that period his condition underwent little change, except that the point of apex-pulsation of the heart was gradually displaced to the left, and the apex-murmur had become harsh.

Re-admitted on the 31st of October, 1866. Four weeks previous to that date, he suffered much fatigue in attendance on his mother in her last illness, and his feet became swollen. A week subsequently he was found one morning in bed, completely paralyzed on the right side, and incapable of uttering a word. He was then put under treatment by the dispensary medical officer, and took mercury till his gums were affected. His actual condition at the date of admittance was the following: Pulse 98, slightly dicrotous, but regular, and of moderately good volume; *rima palpebrarum* slightly larger on right side than on left; pupils equal; tongue protruded to the right, and with vigour; deglutition unaffected. There was complete loss of motor power in the right arm and leg, without impairment of sensibility. The rectum and bladder were unaffected, and vision was not impaired. He was only able to utter the words "yes," "no," "I do," and a

few others, intelligibly, but with great difficulty, in answer to questions put to him.

When asked his name, he tried hard to remember it, corrugating his forehead and eyebrows in the effort, and seemed angry at his failure. When asked was it "Griffin," he at once exclaimed, but indistinctly, "Griffith," which was his name. In reply to a question as to his age, he said, very indistinctly, "a hundred and eighty." I then asked him was he nineteen, and he at once answered "yes."

The action of the heart was strong, and visible in the sixth and seventh intercostal spaces, a short distance inside the nipple-line; at the apex-point the first sound was replaced by a loud *bruit de scie*; this was likewise audible in the axilla, but not at the base, or in the course of the aorta. The second sound was normal at the right base, but accentuated in the pulmonary artery. To have, thrice daily, grs. iij of iodide of potassium, with ℥x of chloric ether, in a tablespoonful of infusion of cinchona.

On the 3rd of November a few vesicles appeared on the left side of the upper lip; on the following day the eruption had extended somewhat over the left cheek, the left inferior eyelid and *ala nasi*, and had become confluent and opaque.

On the 5th, the corresponding parts of the right side of the face were affected by the eruption, which had now extended much on the left side, and partially involved the neck; it consisted of large round pustules, surrounded by a red areola, and clearly resembling those of small-pox, but not like them depressed in the centre. It presented an ecchymatous character. The conjunctivæ were injected, and the eyes were weeping. The patient's general condition was in no degree affected by it; there were no febrile symptoms, and his appetite was rather improved.

13th. Left angle of mouth much retracted and elevated when the patient attempts to cough. A small blister was applied on the left side of the forehead and temple.

On the 19th he was able to walk across the ward, but with a tottering gait, and his articulation was improved.

On the 3rd of December, after a few days of malaise, a fresh crop of the eruption appeared; it commenced where the blister

had been applied, and soon extended over the entire face and neck; it was confluent in several places, forming patches as large as a shilling, and surrounded by bright-red areolæ. There was, however, no fever. He desired to have wine and chicken, and tried to remember the proper words in which to request them; but failing, he became much excited, placed his hand upon his forehead, and passed into a reverie.

On the 11th, he could raise his right arm nine inches from his side, but there was no improvement in articulation. He was soon afterwards discharged.

He was admitted, for the third time, on the 13th of May, 1867, for a fresh eruption of ecthyma on the face and neck. There was then slight improvement in the power of articulating; he was able to utter two words consecutively. His power of comprehension seemed perfect, and he was manifestly irritated at his failure to recall the proper words to express his wants.

A few days preceding the 3rd of June, when he finally left hospital, he had a fainting fit, in which the heart acted tumultuously. At that date the tongue was still protruded to the right, and he walked with a "drag" of the right leg; there was no further improvement in the motor power of the arm, but in both limbs sensibility was perfect.

He died at his home on the 8th, and I failed to obtain permission to examine the body.

I have reported this case at length, owing to the many interesting features which it presents. The paralysis was manifestly of embolic origin, and the ecthymatous eruption was probably a result of the cerebral lesion.

Valvular disease on the right side of the heart, though infrequent relatively to that on the left side,* is by no means absolutely rare. I quite concur with Dr. Stokes in the opinion that lesion of the valves on the right side of the heart is rarely unaccompanied by a similar affection of the corresponding valves on the left side;† but the existence of valvular disease on the left side, by no means implies a corresponding affection on the

* See p. 240.

† *Diseases of the Heart and Aorta*, p. 164.

right side of the heart; the latter is related to the former as an exceptional sequence, not as Dr. Latham believed, in the proportion of 1 in 3.*

Bertin believed that whilst the structural alteration of the valves on the right side is usually fibro-cartilaginous, ossific transformation may be occasionally witnessed; but generally in cases where abnormal communication between the two sides of the heart has existed, the contact of arterial blood having determined a more active inflammatory process.† It is not necessary to remark that ossific change of structure affords no criterion of the activity of antecedent inflammation, nor, indeed, can it be accepted as proof of the occurrence of inflammation in the ordinary sense.

Disease of the pulmonary valves is of extreme rarity. Bertin has published an example‡ in which the orifice of the pulmonary artery was nearly occluded by a horizontal septum, formed by the diseased and agglutinated valves, which presented an aperture of only two and a-half lines in diameter. The foramen ovale was patent. The only physical sign observed was precordial fremitus of systolic rhythm. He mentions a case in which obstruction of the pulmonary artery was caused by a rigid tricuspid valve; a systolic murmur, to the left of the sternum, existed in this case.

According to Hope, systolic murmur in the pulmonary artery is more frequently due to extrinsic causes, than to lesion of the valves.§ The accuracy of this statement must be admitted; but that structural lesion of the pulmonic valves, involving both obstruction and inadequacy, may exist, and that it is actually within the range of positive diagnosis, the clinical records published since the date of Hope's work, conclusively show. He has, himself, given the details of a case (p. 599) in which double murmur, accompanied by fremitus, existed in the site of the pulmonary artery; the systolic murmur was remarkably loud, and

* *London Medical Gazette*, vol. iii., December 27th, 1828.

† *Traité des Maladies du Cœur*, p. 217.

‡ *LVI^e Observation*.

§ *Opus citat.*, p. 76.

transmitted in the course of the pulmonary artery, whilst the diastolic murmur was heard in the line of the right ventricle.

Doctor E. L. Ormerod has published three cases in which the physical signs of disease of the pulmonic valves were identified during life, and on *post mortem* examination, the valves were found extensively disorganized.* These cases are briefly as follows :

1. A woman, aged twenty-one years, was under Mr. Stanley's treatment for syphilis ; a loud systolic murmur was detected, of maximum intensity in a line extending for two to two and a-half inches from the inner portion of the third left intercostal space towards the middle of the left clavicle, but extensively diffused both anteriorly and posteriorly. All the valves were found to be in a healthy state, except those of the pulmonary artery, which were in an advanced state of disease.
2. A man, aged twenty-eight years, under the care of Dr. Bond of Cambridge, had been subject for years to palpitation and dyspnoea on exertion ; a loud and harsh systolic murmur was heard, as mentioned in the preceding case, and shortly before death a diastolic murmur was likewise detected. The pulmonic valves were rolled up, and the orifice barely admitted the passage of a quill. All the other valves were healthy.
3. A man, aged twenty-two years, dropsical, and suffering from dyspnoea, was under Dr. Burrows' care in St. Bartholomew's Hospital. A purring tremor existed over the entire precordium ; the impulse of the heart was strong, and a loud harsh double murmur, most distinct at the junction of the fourth left costal cartilage with the sternum, was likewise detected. A systolic murmur was heard at the ensiform cartilage, and in the line of the right ventricle.

A positive diagnosis of lesion of the pulmonary valves was made in the three foregoing cases.

Doctor Gordon's case,† in which the twofold lesion of the pul-

* *Edinburgh Medical and Surgical Journal*, vol. lxx., 1846.

† *Stokes on Diseases of the Heart*, Case 19, p. 166.

monic valves existed, and was characterized clinically by a double murmur in the site of the pulmonary artery, audible likewise in the back, but not in the arteries of the neck, and unattended with visible pulsation of the arteries, was one of the earliest and most valuable contributions to the literature of this subject.

Doctor Warburton Begbie has published the details of a typical example of the twofold lesion of the pulmonic valves.* A man, aged eighteen years, who had been delicate from childhood, and easily put out of breath, but had not had rheumatism or hæmoptysis, was treated for cough, which was not accompanied with expectoration or lividity. Respiration was feeble; precordial dulness, cardiac impulse, and seat of apex-pulsation were normal. A loud, blowing, systolic murmur was audible over the upper portion of the chest generally; it was not transmitted into the carotids, and was loudest at the left third costal cartilage, near the sternum, where a diastolic murmur of much less intensity, and a thrill, likewise existed. The pulse was 72, regular, and not visible. The precordium was prominent, and the muscles of the left arm and chest were less developed than the corresponding muscles of the right side. The man had been under Dr. Begbie's observation during the last three years of his life; he died of fracture of the skull, resulting from a fall whilst in a state of intoxication. The valves of the pulmonary artery were four in number, and incompetent. All the other valves were normal and healthy. This was, most probably, a case of congenital defect of the valves.

A most interesting example of the same kind, though not verified by autopsy, has been recorded by Professor Lebert of Breslau.† A boy of four years, whose breathing had been embarrassed from the first month after birth, had been cyanosed after the fourteenth month, when he began to walk; at the inner portion of the left third intercostal space a loud systolic bellows-murmur was heard; it was likewise faintly audible in the second and fourth interspaces, but in no other situation. The author refers to five examples of congenital stenosis of the infundibulum of the right ventricle, first described by

* Beale's *Archives of Medicine*, No. 5.

† *Medical Times and Gazette*, January 1st, 1870.

Dittricht, which he had found recorded in medical literature, including one published by himself. In these latter cases he believes the primary disease to have been myocarditis occurring *in utero*; in some cases he believes it to have been of syphilitic origin.

Irrespectively of structural change of the orifice or the valves, other causes of obstruction in the pulmonary artery are occasionally met with. Thus, the pressure of an aneurism; a solid growth within the pericardium, as noticed by Elliotson, or in the anterior mediastinum; or of enlarged bronchial glands, or of a solidified lung, as observed by Latham and Da Costa, may be a cause of obstruction and of systolic murmur in the pulmonary artery.

A case in which an aneurismal pouch, or, at least, one containing blood, situate between the aorta and the pulmonary artery, and causing loud systolic murmur in the latter vessel by pressure, has been recorded by Dr. Flint.*

Doctor G. W. Balfour concludes from clinical observation, that systolic murmur in the left second intercostal space may depend upon two other causes, irrespectively of disease of the pulmonic valves:† (a) the propagation into the left auricular appendix of a mitral reflux murmur of anæmic or organic origin, as first pointed out by Naunyn, or a murmur of mitral narrowing similarly propagated; and (b), the pressure and flattening of the pulmonary artery against the anterior thoracic wall, by the systole of the ventricles, the left lung having been permanently retracted by antecedent disease, as alleged by Quincke; in this case the pulmonary artery would be the seat of the murmur.

In reference to the first mentioned cause, I would only remark that a murmur of mitral reflux should be readily distinguishable from all others by its special localization and diffusion; and that the presystolic murmur of mitral stenosis is rarely audible in the second left intercostal space, and when audible there, it may be identified by its preceding the first sound and the impulse of the heart.

As to the second alleged cause of murmur in this situation, if the theory upon which it is based were valid, visible pulsation

* *Diseases of the Heart*, second edition, p. 243.

† *Medical Times and Gazette*, December 12th, 1874.

in the pulmonic area should be invariably attended by systolic murmur. Yet such is certainly not the case. Indeed, pulsation in the left second interspace, unaccompanied by murmur, is not uncommon in cases of mitral narrowing associated with dilatation and distention of the pulmonary artery and right ventricle, and to this latter condition I believe the pulsation is due.

I have met with an example of partial occlusion of the pulmonary artery beyond the valves, by the pressure and ingrowth of a large cancerous tumor originating in the anterior mediastinum, and involving the lungs.* In this case a loud systolic murmur was audible over the entire precordium, but most intense in the left second interspace near the sternum.

The recorded examples of pulmonic obstructive murmur due to the pressure of a solidified, and preeminently of a tuberculosed, lung, are more numerous. I have repeatedly met with such, but have kept notes of one only. A discharged soldier, suffering from cirrhosis of the liver and ascites, was received into hospital in October, 1874. It was found necessary to perform paracentesis in order to relieve him from embarrassment of respiration, which constituted his most urgent symptom. This operation was done, for the third time, on the 5th of December; and on that day I discovered partial dulness, with moist crepitant râles over the left front, from the nipple to the clavicle; a loud but soft systolic murmur existed in the internal portion of the second intercostal space on the same side. The area of diffusion of this murmur was limited above by the second costal cartilage, below by the lower margin of the third, and on the inside, by the edge of the sternum. Solidification of the left lung, which was manifestly pneumonic, and the development of murmur, were both of recent occurrence in this case, and most probably in the relation of cause and effect; neither could have existed more than a few days, as the chest had been previously examined with negative results in regard both to the lungs and to the heart. The locality and limitation of the murmur left no doubt in my mind that its seat was in the pulmonary artery.

The man died on the 20th of December. The liver was in an advanced stage of cirrhosis, small, dense, and nodulated, the left

* See Case 66, p. 734, and Fig. XLI.

lobe being dwarfed in an extreme degree. The spleen was enlarged, the intestines united by false membrane in a few places, and the peritoneum dense and opaque. The heart was structurally normal; the walls of the left ventricle were thick, and its cavity was obliterated by so-called concentric hypertrophy. The valves were all healthy and competent; the pulmonary artery and the aorta were normal as to size and structure, and they contained no clot. The superior lobe of the left lung was dark in colour, dense, and comparatively dull on percussion; it was non-crepitant, but floated in water. The condition was that of hepatized lung-structure in process of resolution.*

A series of cases of this kind, eight in number, has been published by Dr. Da Costa.† The patients, all young, exhibited indubitable signs of tubercular solidification and softening of the upper portion of the left lung. A loud systolic murmur existed in the pulmonary artery; it was usually soft, but occasionally it was high pitched, and even musical after exertion on the part of the patient, loudest during expiration, and suspended by a full inspiration. This murmur was, in seven of the cases, limited to an area not larger than the bell-end of the stethoscope in the left second intercostal space, close to the sternum; it was not transmitted into the aorta or the carotids, and was accompanied by an accentuated second sound in the pulmonary artery. Dr. Da Costa believes the murmur to have been located in the left branch of the pulmonary artery, and to have been caused by the pressure of the solidified lung.

Murmur in the pulmonary artery may result from thrombosis.

Doctor Gee has reported the case of a young man dying of sun-stroke, in whom he detected a systolic murmur in the pulmonary artery during the last moments of life; the pulse was frequent and feeble, and the temperature in the axilla was 109·5 F. He considered that, owing to the high temperature of the body, a clot had most probably been formed, and impacted in the vessel.‡

Finally, murmurs in the pulmonary artery may be of hæmic

* This case has come under my notice since the paragraphs on the subject at p. 240 were written.

† *American Journal of the Medical Sciences*, new series, vol. xxxvii.

‡ *Gustonian Lectures*, *British Medical Journal*, March 25th, 1871.

origin. When due to this cause, the murmur would necessarily be systolic in rhythm, as in the cases mentioned at p. 240.

Incompetency of the right sigmoid valves would be necessarily attended with diastolic murmur in the pulmonary artery. I have no personal experience of this condition, which constitutes one of the rarest lesions in pathology.

According to Dr. Cheevers, it is seldom witnessed except as the result of injury, or as a congenital defect.*

Doctor Bellingham maintains, that the valves of the pulmonary artery present a cribriform condition almost as frequently as do those of the aorta, the latter being usually affected at the same time; but that, owing to the shortness of the pulmonary artery, the feeble reaction of its walls, and in some degree likewise to the seat of the perforations in the valves, a murmur of reflux is not developed.† I entirely dissent from both these propositions; out of many hundred hearts, exhibiting various forms of disease, which I have examined in the *post mortem* room, I have not met with one example of a cribriform state of the pulmonic valves, whilst the records of medicine show that incompetence of these valves is actually attended with diastolic murmur.

In two out of the three cases published by Dr. Ormerod, and likewise in Dr. Gordon's and in Dr. Begbie's cases (pp. 999 and 1000), diastolic murmur existed in the pulmonary artery, and the valves were found incompetent.

I have already (p. 240) pointed out the diagnostic characteristics of systolic murmur arising at the orifice of the pulmonary artery. I may briefly state here that it is distinguished by the following peculiarities, positive and negative. It is remarkably loud and superficial, as correctly noticed by Hope, limited to a small area having its centre at the sternal extremity of the left second intercostal space, and including the adjacent portion of the second and third costal cartilages, but occasionally audible in the interscapular space. Most frequently it is soft and blowing in quality. Negatively, it is characterized by being inaudible to the right of the middle line of the sternum, in the aorta, and in the carotid arteries. By these negative signs it may be distinguished from a murmur of the same rhythm produced at

* *London Medical Gazette.*

† *Diseases of the Heart*, p. 280.

the orifice of the aorta. From the murmur of an aneurism it must be discriminated by reference to the positive signs of that disease, and especially the circumstance that the murmur of an aneurism, *not* connected with the left extremity of the arch of the aorta, or the descending portion of the vessel, would be traceable, to some extent, towards the right side; whilst, if the aneurism arose from the left portion of the aorta, the effect upon respiratory and percussion-phenomena on the left side of the chest would be sufficiently distinctive.

Inadequacy of the valves at the pulmonic orifice is characterized by a diastolic murmur, most frequently soft and blowing, and transmitted downwards in the line of the right ventricle; that is, towards the ensiform cartilage. From aortic diastolic murmur, that of pulmonary inadequacy may be distinguished, not only by its seat of origin being to the left of the sternum, but likewise, as urged by Drs. Gordon and Begbie, by the absence of visible pulsation in the arteries.

Lesions of the pulmonic valves can rarely be the subject of direct *treatment*, because so seldom the product of acute disease. When detected, defect of these valves from acquired disease is usually so well established, that it is, in regard to the lesion itself, beyond the control of medicine; such is likewise the case when the defect is congenital. The immediate and remote consequences which arise from it, however, namely, surcharge of the general venous system, and defective supply of arterial blood, are in some degree amenable to treatment. This must be conducted on the general principles already pointed out in the discussion of other valvular lesions, and need not be repeated here.

The pulmonary artery is occasionally the subject of *atheromatous change*, and also of *aneurism*. Of the former condition many examples have been published.

Doctor Gerald Yeo has brought under the notice of the Pathological Society of Dublin, a case in which atheroma of the pulmonary artery and its branches throughout both lungs, coincided with constriction of the mitral opening and hypertrophy of the right ventricle.*

* *Proceedings of the Pathological Society*, vol. v., part ii., p. 123.

Doctor Rasmussen has published a list of four cases, in which aneurism of a branch of the pulmonary artery caused fatal hæmoptysis by bursting into a cavity in the lung.* It has been stated by eminent authority that the pulmonary artery is liable to spasm under the operation of certain toxæmic agents.

Doctor George Johnson maintains that, in Asiatic cholera, contraction of the branches of this vessel in the lungs, from the irritation of the cholera poison, is of invariable occurrence in the stage of collapse, and that it explains at once, by reference to the failure of the arterial circulation, suppression of the pulse, depression of temperature, and arrest of secretion; and, on the other hand, by surcharge of the right chambers of the heart and of the systemic veins, that the blue colour of the surface would be accounted for on the same hypothesis.†

Doctor Lauder Brunton has discovered that the dyspnoea attendant on poisoning by *muscarin*, the active principle of the *agaricus muscarius*, is due to contraction of the pulmonary arteries in the lungs, by which the venous side of the heart is engorged, and the arterial side emptied. As the best treatment of mushroom-poisoning, he suggests, after the stomach has been emptied by means of an emetic, that ℥j of the liquor of atropia (B. P.) should be injected hypodermically. This, as he avers, will promptly relax the arterial spasm in the lungs.‡

Disease of the tricuspid valves, although comparatively rare, is of more frequent occurrence than valvular lesions at the pulmonary orifice. The tricuspid valves, though structurally healthy, are, however, in the ultimate stages of nearly all other forms of organic disease of the heart, rendered incompetent by dilatation of the right ventricle. Hence, whilst structural alteration of these valves is exceptional, functional incompetence is of constant occurrence. The signs by which this is indicated have been already discussed (p. 238). Here it is only necessary to remark, that the positive signs of tricuspid inadequacy are usu-

* *Hospitals-Tidende*, Nos. 9 and 13, translated by the late Dr. W. D. Moore. *Edinburgh Medical Journal*, November and December, 1868.

† Pamphlet, 1866.

‡ *British Medical Journal*, November 14th, 1874.

ally limited to the phenomenon of venous palpitation in the neck, where structural alteration of the valve does not likewise exist.

A remarkable example of general venous pulsation in connexion with tricuspid incompetence, has been recorded by Dr. Benson.* It is necessary to observe, that throbbing of the carotid arteries, in connexion with inadequacy of the aortic valves, or with extreme debility of the left ventricle, if associated, as it frequently is, with congestion of the right chambers of the heart, may be readily mistaken for intrinsic pulsation of the jugular veins. The distinction may, however, be easily made, by pressing the finger lightly upon the distended vein above the clavicle. The effect of this will be to arrest the pulsation, if it be due to reflux from the auricle; whereas, if the pulsation be arterial, this expedient will have no result. It may be further remarked, that the true jugular pulsation is partially arrested by collapse of the veins during *inspiration*, whilst it is exaggerated at the acme of *expiration*. The carotid pulse is in no degree influenced by respiration.

The absence of murmur in cases of simple functional inadequacy may be explained by reference to the debility of the right ventricle, which accompanies dilatation of that chamber. Murmur may, however, be developed in such cases by the engagement of a vibrating shred of fibrin in the orifice. Inadequacy of the valves may be likewise produced by a band of fibrin entangled in the tendinous chords of one or more of the segments, and attached to the wall of the ventricle; but it is usually unaccompanied by murmur.

The valve-segments are, however, occasionally thickened and tuberculated in a degree sufficient to render them inadequate, by antecedent endocarditis, generally rheumatic, and coinciding with a similar affection on the left side of the heart.

Acute inadequacy may likewise result from rupture of the valve, the papillary muscles, or the tendinous chords. Examples of the two former lesions have been recorded by Dr. Da Costa,† and a typical case of the latter accident has been published by

* *Dublin Journal of Medical Science*, first series, vol. viii., 1836.

† *Toner Lectures*, No. III., Washington, May, 1874.

the late Dr. Todd.* The symptoms and signs by which it is characterized are sudden dyspnoea, with constrictive pain in the chest, and tendency to syncope. At a later period, engorgement of the liver and kidneys and general dropsy supervene.

Doctor Todd noted repeated hæmatemesis as a symptom in his case, and this he attributes to acute engorgement of the portal system, through congestion of the right auricle. In all these cases the accident has been the result of a sudden wrench or strain, and the injured structures had been presumably unsound when laceration occurred.

Murmur of systolic rhythm and of low pitch is a necessary sign in such cases, because the ventricle, as yet undilated, possesses sufficient contractile force to produce it. The seat of maximum intensity of a tricuspid regurgitant murmur is at the base of the xyphoid cartilage, or slightly to the left of that point; and the line and extent of transmission is upwards and to the right, for a distance of not more than one to two inches.

The limitation of transmission just adverted to constitutes the principal element of differential diagnosis, as between tricuspid and aortic murmurs of systolic rhythm; a further but less constant element is the quality of the murmur, which, when aortic, is usually harsh, whilst it is soft and faint when of tricuspid origin, except in those cases where a vibrating thrombus may have modified its character.

If, with a murmur of the character and area just described, jugular pulsation should be found to coincide, a positive diagnosis of tricuspid reflux may be confidently made.

The phenomenon of venous pulsation in the neck has been already discussed (p. 571). I need only remark here, that it is an infallible indication of tricuspid inadequacy, and, whether accompanied or not by murmur, if the sources of mistaken identity mentioned at p. 1007 be avoided, it may be accepted as proof of tricuspid regurgitation.

Valvular obstruction at the tricuspid orifice is usually associated with a similar lesion of the mitral valve, and may be conveniently discussed in that connexion.

* *Dublin Journal of Medical Science*, new series, vol. v. See also a case by Mr. Squire, *Transactions of London Pathological Society*, vol. xvi., 1864.

Coincident obstructive disease at both auriculo-ventricular orifices is of rare occurrence, yet a few well authenticated examples of it have been recorded. I have myself met with three such examples. Still more rare has been the identification of the twofold lesion during life. Even this, however, has been accomplished in at least three instances.

Narrowing of the tricuspid orifice has been associated with a similar condition of the mitral opening in every recorded example of the lesion that has come under my notice, with the single exception of one published by Bertin.* It is further to be noticed that in the great majority of the cases, the mitral lesion has been in excess, and in point of time, manifestly in advance of the tricuspid.

Bertin has recorded an example of the twofold lesion which came under his notice in 1814.† The physical signs are not mentioned, nor was a diagnosis attempted.

I find recorded in the Transactions of the London and Dublin Pathological Societies, only nine examples of this lesion, three in the former, and six in the latter. The cases which have been published in the United Kingdom and America, arranged chronologically, are briefly as follows.

In February, 1848, Doctor Quain exhibited the heart of a man, aged thirty-seven, who had acute rheumatism at the age of fifteen years. The most prominent symptoms were cough and hæmoptysis: death occurred by syncope. The lungs were found tuberculous and excavated. The pericardium was adherent to the heart, and, in the false membrane, and likewise in some degree imbedded in the substance of the heart, a calcareous mass was found. The heart weighed fourteen ounces. The segments of the tricuspid valve were thick and opaque, and united for one-third of their extent, the orifice left admitting only the points of two small fingers. The mitral valve and the corresponding chordæ tendinæ were thickened. The mitral orifice was contracted so as to admit only two fingers, and on the auricular surface of the valve were two osteoid nodules. The aortic

* *Traité des Maladies du Cœur, LVII^e Observation.*

† *Opus citat., XLIX^e Observation.*

valves were thickened, and the edge of one of them was everted. No diagnosis of the cardiac lesions was made.*

In January, 1850, Doctor Pollock reported the case of a woman, aged thirty-eight, who suffered from great dyspnoea, with lividity and general dropsy. There was a systolic murmur, loudest "towards" the apex, but also audible at the base; and over the sternum, at the level of the third rib, a diastolic murmur was likewise heard. She never had rheumatism. The right auricle was found greatly dilated and thickened. The tricuspid valves were united and thickened, forming a diaphragm provided with a central aperture, which admitted only one finger. The right ventricle was reduced in size. The aortic valves were disorganized and incompetent, and the aorta was atheromatous. The mitral valves were thickened, and the orifice so contracted as to admit only one finger. No special diagnosis was made.†

In January, 1852, Dr. Pye Smith submitted the following case: A woman, aged thirty-seven, who never had rheumatism, suffered from dyspnoea, palpitation, and anasarca. The impulse of the heart was strong. A rough systolic murmur, loudest at the apex, was heard, and next, a harsh diastolic apex-murmur. The heart weighed nine ounces. The right auricle was thickened, and enormously dilated. The right ventricle was dilated, and the tricuspid orifice was much contracted. The mitral orifice was greatly contracted, rough, and calcareous, and the attached tendinous chords were thickened. The aortic valves were thick, rough, and inadequate. The diagnosis of auriculo-ventricular narrowing was not made.‡

In 1862 Professor Gairdner made an all but positive diagnosis of tricuspid narrowing in the person of a man, aged twenty years, who suffered no inconvenience from his disease, except the slight amount arising from pulsation of the jugular veins. A presystolic ("auricular-systolic") murmur was detected, and upon this the diagnosis was based.§ The patient died in 1872, and the heart was exhibited by Dr. Gairdner at one of the sectional

* *Transactions of the Pathological Society of London*, vol. i.

† *Ibid.*, vol. ii.

‡ *Ibid.*, vol. iii.

§ *Clinical Medicine*, 1862, p. 602.

meetings of the British Medical Association in London in the following year. It confirmed his diagnosis, in so far as that an obstacle to the entrance of blood actually existed at the tricuspid orifice; this, however, arose, not from stenosis or from valvular lesion of the orifice, but from the presence of a globular tumor which was attached by a pedicle within the auricle, and floated down upon the orifice after the manner of a ball-valve. This extremely interesting and unique example of disease at the tricuspid opening does not strictly belong to the category of obstructive lesions of the tricuspid valve, although closely allied with them by symptoms and physical signs; the absence of coincident disease at the mitral orifice constitutes, therefore, no exception to the statement above made, in regard to the all but constant association of mitral with tricuspid obstruction.

In December, 1862, Dr. Stokes exhibited before the Pathological Society of Dublin, the heart of a female, aged twenty. The patient never had rheumatism. There had been great variability in the sounds of the heart, and intermittent cyanosis. She was subject to headache and palpitation. A systolic murmur, loudest at the right side of the heart, was occasionally present; it was, however, frequently absent, and a diastolic murmur was occasionally heard. The erect posture increased in an especial manner the variability of the murmur. A month before the patient's death, the murmur was often observed to be presystolic. The carotid arteries pulsated visibly, especially that of the right side, and most strongly in the recumbent posture, and there was a venous thrill at the root of the neck. A murmur was likewise audible in the carotid arteries, and in the left scapular region. At the junction of the third costal cartilage with the sternum, on the right side, a prolonged systolic murmur existed, which was followed by the sounds of the heart. Death took place suddenly by rupture of an intracranial aneurism; and whilst the patient was *in articulo mortis*, a substitutive diastolic murmur was heard. The aortic valves were utterly disorganized; the mitral valves were also diseased, and the mitral orifice was contracted to the diameter of 5×5 lines. The tricuspid valves were partially coherent, reducing the diameter of the tricuspid orifice to 7×6 lines. The heart weighed twenty-five ounces.

In the foregoing case, an approximate diagnosis was made; a presystolic murmur, which was localized at the right side of the heart, was identified.*

In April, 1867, Dr. James Little exhibited before the Pathological Society of Dublin the heart of a female, aged twenty-three, who for thirteen years previously had been out of health, and for the last six months especially had suffered from an affection of the chest. The feet were swollen, and the patient had cough, accompanied with dyspnoea and lividity. Evidence of pulmonary emphysema with bronchitis existed. Owing to the extreme respiratory distress, a satisfactory examination of the chest was not made, and no murmur was detected. The right auricle was found to be dilated and thickened, and the right auriculo-ventricular opening reduced to the diameter of the point of the index finger. The left auriculo-ventricular opening was likewise much contracted, but not to an unequal degree with the right. The aortic valves were slightly incompetent. No diagnosis as to the state of the heart was attempted in this case.†

Doctor Balthazar Foster, in the year 1868, observed and reported a case in which he had made the positive diagnosis of tricuspid narrowing.‡ A murmur of presystolic rhythm was detected at the base of the ensiform cartilage; it was, however, loudest to the right of that point, was propagated faintly to the base, but scarcely, if at all, to the apex of the heart. There was pulsation of the jugular veins, recognised as of presystolic rhythm, and fremitus accompanied the murmur at the ensiform cartilage. On *post mortem* examination, the tricuspid orifice was found to be greatly contracted; the mitral opening was likewise, but in a less degree, narrowed.

Doctor Foster remarks that in cases of tricuspid stenosis general venous congestion is greatly in excess of pulmonic. Such is certainly the case, and it constitutes an accessory symptom of much value.

In July, 1869, an example of the twofold lesion came under my notice. A presystolic and a systolic murmur existed at the

* *Proceedings of the Pathological Society of Dublin*, new series, vol. ii., part i.

† *Ibid.*, vol. iii., part ii.

‡ *Clinical Medicine*, 1874, p. 324.

apex, and the diagnosis of mitral obstruction and regurgitation was made. No murmur was detected elsewhere, and tricuspid obstruction was not suspected. After death, extreme mitral narrowing was found to exist; the tricuspid orifice was likewise partially contracted.*

Doctor Cryan exhibited a remarkable example of the double lesion in February, 1870. The subject was a female, aged twenty-five. Dr. Cryan made the diagnosis of mitral obstruction and regurgitation; he also diagnosed tricuspid regurgitation, but did not identify tricuspid obstruction. The following remark, which is of the utmost importance in regard to diagnosis, was made by Dr. Cryan: "We could distinguish two seats or centres of murmur, one at the left apex of the heart, and the other at the junction of the fifth right costal cartilage with the sternum."†

Doctor Flint, whilst declaring that a tricuspid direct murmur is "one of the rarest curiosities of medical experience," states that he has met with one example of it. The case was of a complex character, including obstruction at the mitral, tricuspid, and aortic orifice. Doctor Smyth, of Brooklyn, by whom the patient, a young woman, was ordinarily treated, made a presumptive diagnosis of double auriculo-ventricular narrowing, from having heard a presystolic murmur "not only around the apex, but at, and to the right of, the ensiform cartilage."‡

In January, 1871, I submitted to the Pathological Society of Dublin the details of another case of this kind. The patient was a man, aged twenty-three. There was advanced disorganization of the aortic and the mitral valves, involving both obstruction and regurgitation at the respective orifices, all of which had been diagnosed. But, in addition, there was tricuspid narrowing, which had not been recognized.

In reference to this case, I remarked that, "diagnostically the case is of considerable interest. It is perfectly novel to me, and, with the light it affords, I should now have no difficulty in diagnosing, in a similar case, the existence of constriction of the

* See Case 110, p. 939.

† *Proceedings of Dublin Pathological Society*, new series, vol. iv., part ii., p. 144.

‡ *A Practical Treatise on the Diseases of the Heart*, second edition, p. 240.

two auriculo-ventricular openings. The point on which the diagnosis turns is this, that whereas the murmur of mitral constriction is always at the apex of the heart, and, in the great majority of cases, strictly limited to the area of the mitral opening, in this case a murmur of the same rhythm was audible to the left of the sternum. Between these two points there was a portion of the chest over which no murmur was distinctly audible.*"

In April, 1872, Dr. Cryan submitted the particulars of another case in which, from the presence of systolic murmur at the apex, and turgescence, with systolic pulsation of the jugular veins, the diagnosis was made, of "mitral insufficiency, combined, *in all probability*, with contraction of the mitral orifice; tricuspid regurgitation, due probably to dilatation of the right auriculo-ventricular opening; hypertrophy, and dilated heart." Both auriculo-ventricular orifices were found to be much contracted, the left in a greater degree than the right; the heart was globular, and weighed eleven ounces.†

The report of the following case, the seventh recorded in Dublin, was communicated by me to the Medical Society of the King and Queen's College of Physicians, in March, 1874. The morbid specimen was, at the same time, exhibited to the members of the Society. A woman, aged twenty-five, for many years out of health, suffered from cough, hæmoptysis, great dyspnœa, and œdema of the lower limbs. There was congestion of the face and neck, and great turgescence of the jugular veins. A presystolic murmur was detected at the apex of the heart, which pulsated to the left of the nipple-line. A murmur of the same rhythm, but harsher, was heard near the left margin of the sternum, in the fourth intercostal space. At a point intermediate to these two seats of murmur, neither was distinctly audible. The diagnosis of both mitral and tricuspid obstruction was made, and confirmed by the autopsy.‡

In November, 1872, a man, aged twenty-five, the subject of double auriculo-ventricular contraction, was admitted into hos-

* *Proceedings of the Dublin Pathological Society*, vol. iv., part iii.

† *Ibid.*, vol. v., part i.

‡ See Case 116, p. 956.

pital for me. He came from a distant part of the country to obtain relief from palpitation and weakness. He had never had rheumatism, and his health had been good up to six months previously. Since then, he had repeatedly spat blood.

There was no œdema, cough, or dyspnoea, and no engorgement of the cervical veins. He frequently sighed, and declared he experienced relief from the act. The pulse, in the radial artery, could not be registered, owing to its extreme weakness and irregularity. The pulsations of the heart, which were likewise irregular, numbered 120 in the minute. The apex-pulsation was felt in the nipple-line and fifth interspace. At this point a presystolic fremitus was felt, and a loud and harsh presystolic murmur was heard. This latter was loudest at two points; namely, at the apex, and two inches to the inner side of, and somewhat below the left nipple. The second sound was not double or accentuated.

The man's health improved under treatment with iron, digitalis, and strychnine; but the sighing continued, and the physical signs were as above described at the date of his discharge, a fortnight after admittance. I have not since heard of him. The diagnosis of the double lesion was made from the existence of two distinct seats of murmur, accompanied with fremitus.*

A fifth example of auriculo-ventricular narrowing, on both sides of the heart, came under my notice in July, 1874. The patient was a woman, aged fifty-six, suffering from dyspnoea and œdema, and subject to hæmoptysis. The face and neck were congested, and more so after exertion; they were usually swollen in the morning. A presystolic murmur existed at the apex, and a murmur of the same rhythm was heard in the fourth interspace, near the left margin of the sternum.† The diagnosis was made, as above mentioned; but the opportunity for testing its accuracy by *post mortem* examination, was happily not afforded, as the poor woman improved somewhat under treatment, and left hospital after a fortnight's residence, to resume her duties as a midwifery nurse.

It would seem that Hope had not met with an example of

* See Case 52 in Table XI.

† See Case 15 in Table XII.

tricuspid narrowing. M. Aran has declared that he himself had not.*

In 1868, M. Raynaud questioned the possibility of making the differential diagnosis of tricuspid obstruction. He says: "As to morbid sounds of diastolic and presystolic rhythm, we are not convinced that they can be referred, with certainty, to the right side of the heart, and we even doubt that the right auricle possesses sufficient force to produce a murmur immediately preceding the ventricular systole." He adds: "What well-informed practitioner, then, will be bold enough to say, 'This belongs to the right, and that to the left heart?'"†

Amongst the preceding cases will be found at least three, which supply an affirmative answer to this query. These three cases establish, beyond question, the practicability of distinguishing between mitral and tricuspid presystolic murmur.

In reference to treatment, I would only remark that, in addition to the measures recommended in cases of mitral obstruction, all of which are applicable to the double lesion, the abstraction of a few ounces of blood from the arm would afford at least temporary relief to the state of high venous tension which exists in the latter cases.

Disease of the coronary arteries, consisting in atheromatous change or calcification of their coats, has been met with most frequently in connexion with similar changes at the root of the aorta, involving the valves, the walls of the vessel, or both. It has been likewise witnessed very frequently in cases of fatty degeneration of the heart.

Doctor Quain found the coronary arteries more or less altered and obstructed in 13 out of 33 cases of fatty degeneration recorded by him, and in 1 case (26, series I.), the branch of supply from the coronary artery to the *only* portion of the heart which exhibited fatty metamorphosis, was completely calcified.‡ The connexion between disease of the coronary arteries and fatty degeneration of the substance of the heart, may be readily ex-

* *Archives Générales de Médecine*, tom. xv°, 1842.

† *Dictionnaire de Médecine et de Chirurgie*, tom. viii°, p. 643.

‡ *Medico-Chirurgical Transactions*, vol. xxxiii.

plained by reference to the impairment of cardiac nutrition to which disease of these vessels must lead, on the assumption, which cannot be disputed, that fatty transformation of muscle is an atrophic change. Similar results may, however, follow from simple obstruction of the coronary arteries at their origin, by calcareous deposition in the sinuses of Valsalva. I think it may be assumed, even in the absence of disease of the coronary arteries, or occlusion of their orifices, that fatty disease of the heart is the direct result of a feeble circulation through these vessels, in cases of primary dilatation and atheroma of the first portion of the aorta. In such cases the coronary circulation, which depends mainly, if not entirely, upon the recoil of the aorta, fails, owing to the rigid and inelastic state of the walls of that vessel.

Thrombosis of the coronary arteries may occur in cases of parietal disease of these vessels, and become the cause of acute fatty transformation of the heart. Calcification of these vessels has been regarded as the essential cause of angina pectoris; but this doctrine is too exclusive, and is not admitted by modern pathologists.

CHAPTER VIII.

FORMATION AND MIGRATION OF BLOOD-CLOTS.

THROMBOSIS of the heart, or the formation during life of clots or so-called polypi within its chambers, consisting of separated fibrin and blood corpuscles in a more or less altered condition, may occur as a consequence of: (*a*) The admixture with the blood of foreign matters in a state of minute division, or of organic fluids in process of septic change;* (*b*) Hyperinosis, as in acute rheumatism; (*c*) High pyrexia, as in the early stage of scarlatina;† (*d*) Retention of excreta, as in chronic renal disease;‡ (*e*) Pyæmia; (*f*) Leukæmia; and (*g*) Protracted death struggle in cardiac, renal, or pulmonary disease.§

Doctor Bristowe concludes that "sex and age have no obvious influence on the formation of cardiac thrombi; that they occur in all the cavities of the heart, but most frequently in the left ventricle, and least frequently in the left auricle; that they affect, with scarcely an exception, those parts of the cavities which are out of the direct line of the circulation, and are always fixed by being entangled amongst the inequalities of surface; that they are met with, almost exclusively, in those forms of disease in which the process of death is, as a rule, protracted; that their arrangement, form, and microscopic characters seem to show that (excluding one or two unusual cases) they consist merely of blood which has coagulated during life, and has undergone those changes alone which clots elsewhere in the living body are liable to undergo; that they are the result of spontaneous coagulation of blood in the heart's cavities, at a variable period anterior to death, but occurring at a time at which the

* Mr. Savory, *St. Bartholomew's Hospital Reports*, vol. i.

† Dr. John Harley, *Medico-Chirurgical Transactions*, vol. iv.

‡ Dr. Bristowe, *Transactions of Pathological Society of London*, vol. vii., and Dr. John Ogle, *ibid.*, vol. xiv.

§ Bristowe, *loco citat.*

patient is in a moribund condition, or in a condition of threatening dissolution.*

These masses consist mainly of fibrin more or less completely separated from the other constituents of the blood, and exhibiting various shapes, according to the size to which they have attained, and the positions which they occupy. Most frequently they are smooth on the surface, and of a light buff tint, but occasionally the colour is nearly white. When small, they may be ribbon-shaped, the band extending from the auricular appendix, where it is firmly attached to the pectinate columns, or to a dense mass of the same structure there impacted, through the auriculo-ventricular opening into the ventricle, and sometimes from the ventricle into the pulmonary artery or the aorta. This band not unfrequently renders the respective valves incompetent by being entangled in their edges, or in the tendinous chords; it occasionally, though rarely, takes origin from the apex of either ventricle, proceeding thence towards the arterial orifice, or into the artery. Thrombi of this figure usually coincide with coloured coagulum, and are blood-stained on the surface; but their structure is fibrinous and uniform throughout. Occasionally the auricle or ventricle on either side of the heart is all but completely filled by a large thrombus, which has assumed the shape of the cavity, forming a perfect but reduced cast of it, and leaving only a narrow interspace on the surface through which an imperfect circulation is still maintained.

I have seen both the right chambers and the tricuspid orifice occupied in this manner, whilst the left cavities contained some detached clots and flakes. Massive thrombi of this kind consist of a shell of decolorized fibrin, from half a line to a line thick, and a soft and grumous parenchyma, composed of the *débris* of fibrin and blood corpuscles, and usually tinted red.

Doctor John Ogle has found in these bodies "amorphous molecular and fatty material, imperfect cellular, nuclear, and fibrillary forms, with compound granular corpuscles."† Within a short time, as a consequence of disintegration and putrefactive change, the contents may assume the appearance and the consistence of pus; and, as Dr. Ogle surmises, they may be diffused

* *Loco citat.*

† *Loco citat.*

in the circulation by rupture of the cyst, and so give rise to symptoms of pyæmia. That the contents of cystic thrombi within the heart are never truly purulent, may be now regarded as proven.

Smaller bodies, composed of fibrin in process of change, and presenting various figures, are constantly found in the heart, even where no obvious symptoms have been attributable to their presence; such are flakes and bands of fibrin attached to the papillary muscles and tendinous chords, and floating loosely in the cavity; small irregular bodies of a solid but friable structure, impacted in the recesses between the fleshy columns; nodular deposits upon the valves; and pedunculated globular masses, from the size of a pea to that of a walnut, suspended from some portion of the wall, as described by Laennec.* The thrombus may consist in a thick lining of precipitated fibrin, extending throughout both the right chambers, and forming a hollow cast of them, through which the blood had circulated.†

I believe the right chambers of the heart are by many degrees more frequently the seat of thrombosis than the left chambers; a difference for which the less degree of muscular development, and the greater liability to stasis on the right, as compared with the left side of the heart, will supply an adequate explanation. Of the 44 fatal cases of valvular lesion given in Table X. (p. 815), 24 exhibited thrombosis on the right side of the heart, and 12, thrombosis on both sides. In no instance was the left side alone thrombosed.

I have witnessed cardiac thrombosis more frequently in connexion with dilatation and debility of the right chambers of the heart than with any other condition. Obstruction on the left side of the heart, and pulmonary emphysema with chronic bronchitis, issuing in dilatation and distention of the right chambers, have furnished the greatest number of examples. The two essential conditions for precipitation of fibrin from the circulating blood are herein supplied; namely, stasis, and a rough surface.

Irrespectively of symptoms, most valuable evidence of the

* "*Végétations Globuleuses*," *Traité d'Ausculation Médiate*, tom. ii., p. 630.

† See Case 110, p. 939.

ante mortem formation of these bodies is occasionally furnished by their configuration and surface marking. An impression of the aortic valves, in a state of partial closure, has been found by Sir Dominic Corrigan upon a thrombus extending from the left ventricle into the aorta.*

I have frequently observed a similar condition of the clot engaging the aortic orifice, and likewise the mitral and tricuspid openings.

The *symptoms* of cardiac thrombosis are, in many cases, sufficiently pronounced to warrant a positive diagnosis. It must, however, be added, that in the majority of instances the evidence available during life will admit of a presumptive diagnosis only; this observation applies to the cases in which bands or small masses, not implicating the valves or the orifices, have been formed.

Extreme thrombosis of the right chambers may be confidently diagnosed from the following symptoms occurring in a case of chronic pulmonary or cardiac disease: Reduction in volume, and increase in the rate of the pulse; faintness; orthopnoea, characterized by a struggle for breath, without difficulty of breathing (air freely enters the lungs, but it does not satisfy the *besoin de respirer*); tumultuous and irregular action, with occasional intermissions of the heart, feeble impulse according to Richardson,† and partial or complete suppression of the sounds on the right side. The area of precordial dulness is extended to the right; the respiratory sound is full, and even sonorous. The nares are dilated, the chest heaves with inspiration, whilst the patient is agitated, moans, and craves for air;‡ the veins of the neck are turgid, the features anxious and livid, and the surface cold, and often humid with perspiration.§

The symptoms of clot on the left side of the heart are of a less distinctive character. Richardson avers that the sounds are faint or suppressed on the left, though audible on the right side, whilst the impulse is heaving and tumultuous. I am not in a

* *Proceedings of the Pathological Society of Dublin*, new series, vol. ii., part i.

† *Medical Times and Gazette*, November 21st, 1868.

‡ "Summæ anxietates" of Van Sweiten.

§ See Case 106, p. 927; also Cases 107, p. 932; and 111, p. 942.

position either to confirm or deny the accuracy of this observation. The weakness of the radial pulse, however, contrasts with the strength of the cardiac impulse, whilst the signs of pulmonary congestion are well pronounced, and often associated with hæmoptysis.

On either side of the heart, a small and floating thrombus, engaged in one of the orifices, may give rise to a systolic murmur of a more or less musical character.

The *treatment* of thrombosis of the heart must be preventive rather than curative. Whilst we cannot hope to effect solution of the clot already formed, we may succeed in preventing its further growth by medicine. Alkalies afford the best, although a doubtful prospect of accomplishing the latter object.

Doctor Richardson proposes to give, at short intervals, $\mathfrak{M}\text{x}$ doses of the *liquor ammoniæ*, in an ounce of water; whilst Professor Gerhardt prefers bicarbonate of soda, which he administers by inhalation, as a more direct mode of introducing it into the heart. The solution used by Gerhardt contains from a-half to one-and-a-half per cent. of the salt, and the results, as reported by him, are encouraging.*

Thrombosis of the arteries and veins is of rare occurrence in the absence of local inflammation of the walls of the vessels; and in these exceptional instances it is usually secondary to an impacted embolus.

Doctor Liddel, however, thinks that arterial thrombosis may result from an excess of fibrin, or of white corpuscles, in the blood; or from atheromatous or calcareous change in the coats of the vessels.†

I have occasionally seen a deposit of fibrin upon the internal surface of the aorta, where roughened and fissured in the process of atheromatous transformation; but in most instances there has been local dilatation of the vessel in the seat of the deposit.

Thrombosis of the veins usually depends upon phlebitis in the seat of the clot. Uterine phlebitis, consecutive to parturition, is the most frequent cause of it; hence it is usually exhibited in the

* *Deutsches Archiv. für Klinische Medicin*, vol. v., p. 207, summarized in *The Dublin Quarterly Journal of Medical Science*, May, 1869, p. 421.

† *American Journal of Medical Science*, January, 1873.

femoral and internal saphena veins. This is true, not only of the *post partum* form of venous thrombosis, but likewise of this affection arising from other causes, owing to the tendency to stasis in the veins of the lower limbs, as compared with the other portions of the body. These vessels are unfavourably circumstanced in regard to their circulation, not only by having to sustain the weight of the column of blood in the iliac veins and the inferior cava, and to propel their contents against gravity in the upright posture, but also by the casual obstruction to which they are liable from pressure within the abdomen, from stasis in the lungs, or in the right chambers of the heart.

The cerebral sinuses and veins are, next after the veins of the lower extremities, the most liable to thrombosis. This arises, most probably, from the slowness and equability of the circulation, which results from the absence of direct atmospheric pressure within the cranium.

The gravity of venous thrombosis depends upon the danger of consecutive embolism, the seat of which, when the systemic veins are engaged, is the right side of the heart and the pulmonary artery.

Phlebitic thrombosis may arise, not only from mechanical injury to the veins, and the direct introduction of septic poisons into those vessels, as occurs in the female after parturition, but likewise from the operation of certain morbid agents developed in the blood itself, or from changes of its constitution.

Doctor Tuckwell has recently published three cases of phlebitis, followed by embolism of the pulmonary artery, in gouty subjects, produced, as he believes, by the operation of the morbid element of gout in the blood.* Sudden death occurred in two of these cases; in one of the latter the internal saphena and the femoral veins of one limb were found to have been the primary seat of inflammation and coagulation; the man, who was gouty, was convalescent when he imprudently got out of bed, and, "in the act of bending his thigh to get into bed, he broke off a large piece of the clot in the femoral vein, which rushed into the left pulmonary artery, and suddenly deprived him of the use of his

* *St. Bartholomew's Hospital Reports*, vol. x. 1871.

left lung," the right lung having been previously and universally infarcted, as shown by the autopsy. The femoral vein, the right ventricle of the heart, and the left pulmonary artery, were occupied by separate portions of a large cylinder of solid fibrin, the broken ends of which accurately fitted to one another. In the third case, typhoid or pyæmic symptoms were developed, which were supposed to have arisen from blood-poisoning by disintegration of the clot; the patient ultimately recovered.

A case of pulmonary embolism, consecutive to phlebitis of the internal saphena in a pregnant female, has been published by Mr. Brudenel Carter. Improvement did not take place, as usual, after confinement, and the patient soon after died quite suddenly. A clot was found in the pulmonary artery.*

Doctor Tuckwell† also furnishes, in the same article, the particulars of two cases of chlorotic venous thrombosis, one in the lower extremities, and the other in the cerebral veins and the sinuses of the dura mater. In the latter, which was suddenly fatal, nearly all the sinuses and veins of the right hemisphere of the cerebrum were found full of coagulated blood; extravasation and softening, to a slight extent, had likewise taken place.

A case similar to that last mentioned has been reported by Dr. Andrew.‡ Severe and persistent headache, followed by coma, were the chief symptoms in both these cases.

I have met with examples of venous thrombosis of the lower limbs in the last stages of pulmonary phthisis. The following is an example of this kind. John B., aged twenty-seven, a coal heaver, was received into hospital in May, 1869. He had been intemperate, and five months previously he was first taken ill. When admitted, he was suffering from dyspnoea, with cough and expectoration. Both lungs exhibited signs of extensive tubercular softening. A few days after admittance, he complained of pain in the calf of the right leg, the limb became swollen, and likewise tender in the course of the femoral vein. On the 9th the entire limb was livid, greatly swollen and very tender, the femoral vein in Scarpa's triangle, was rigid

* *Medical Press and Circular*, April 17th, 1872.

† *Loco citat.*

‡ *Reports of Pathological Society of London*, vol. xvi.

and knotty, whilst the femoral artery pulsed strongly in the same situation ; the temperature of the limb was not depressed.

The man died soon afterwards, but without having exhibited symptoms of embolism. The right femoral vein was found full of decolorized and solid fibrin, which was firmly attached to the coats of the vessel to the extent of two inches. No other parts were examined. In all such cases, Dr. Tuckwell very properly insists upon the necessity for rest and immobility in the recumbent posture ; sudden flexion of the limb should be especially avoided.

Embolism, or the migration of clots within the blood-vessels, is a subject which has been recently studied with great earnestness, and has yielded most remarkable results. Many cases of sudden death, and still more numerous examples of sudden hemiplegia without loss of consciousness, and of instantaneous loss of vision in one eye without previous disease of the organ, which, under the pathology of thirty years ago would have been totally inexplicable, may be now demonstrably traced to this accident.

Professor Trousseau* has assigned to Legroux the merit of having been the first to enunciate this doctrine. To him is due the honour of its revival only ; for, as shown by Dr. Walshe, Van Swieten had anticipated him by more than half a century. After the observations of Legroux (1827), came those of Allibert (1828), Louis and Lediberder (1837), and Baron (1838).† Virchow has investigated the subject with his customary exhaustiveness,‡ but the late Dr. Kirkes was the first to establish conclusively the connexion between valvular disease of the heart and embolism.§ He published, in 1852, three cases of suddenly developed hemiplegia consecutive to cardiac disease. In all three the lesion was valvular ; mitral alone in two ; mitral aortic and tricuspid in one. The valves, in each of these instances, were studded with fibrinous deposits, in the form of vegetations or flakes. The middle cerebral artery of either side was found plugged with

* *Clinical Lectures*, Sydenham Society's edition, vol. iii., p. 414.

† Walshe, *Diseases of the Heart*, fourth edition, p. 461.

‡ *Froriep's Neue Notizen*, 1846, see p. 910.

§ *Medico-Chirurgical Transactions*, vol. xxxv., 1852.

fragments of fibrin in all these cases, and the corpus striatum and adjacent portions of the brain were softened. The renal and splenic arteries were likewise plugged. In a fourth case, a sacular aneurism of the left ventricle existed; the sac was filled with laminated fibrin, the splenic artery was plugged, and the spleen and kidneys exhibited patches of capillary infarction.

Doctor Bristowe has given the particulars of seven cases of plugging of the cerebral arteries, in two of which it was due to detachment of fibrin from diseased cardiac valves.*

Doctor Hughlings Jackson has published several examples of hemiplegia in connexion with valvular disease of the heart, in which he had diagnosed cerebral embolism.†

Of 37 cases under Dr. Hammond's care, in which cerebral embolism had been diagnosed, organic disease of the heart existed in all except one; and in 7 of these cases, in which the body was examined after death, the left middle cerebral artery was found to be the seat of an embolus.‡

From the records of the Pathological Institute of Berlin (1868-'71), it appears that emboli existed in 29 per cent. of all the cases of endocarditis; in two or three instances they originated on the right side of the heart, and produced infarction and abscess in the lungs, chiefly in the inferior lobes; in 26 per cent. of the cases the emboli were derived from the left side of the heart; and of these, the mitral valves alone were found to have been their source in the proportion of 87 per cent., and both the mitral and the aortic valves in the proportion of 49 per cent. The kidneys were the seat of embolism in the proportion of 75 per cent.; the spleen, 51 per cent.; the brain, 20 per cent.; the intestinal tract and liver, 7 per cent.; the skin, 5 per cent.; the spinal cord, 3 per cent.; and the thyroid body and the eye in a somewhat less proportion.§

Doctor Olaf Page, of Valparaiso, has published the case of a young man, the subject of organic disease at the aortic orifice, in whom sudden loss of vision was found, on *post mortem* examina-

* *Transactions of Pathological Society of London*, vol. x.

† *Clinical Lectures and Reports of the London Hospital*, vol. i., 1864.

‡ *A Treatise on the Diseases of the Nervous System*, 1871.

§ *Edinburgh Medical Journal*, July, 1873.

tion, to have been due to embolism of the retinal artery; the left middle cerebral artery was likewise plugged.*

An example of embolism of the right axillary artery, consecutive to thrombosis of the left auricle or ventricle in a chlorotic female, free from organic disease of the heart, is given by Dr. Tuckwell;† gangrene of the fingers took place, but the patient recovered.

Amongst the cases of valvular disease recorded in the preceding pages of this work, 136 in number, 13 examples of hemiplegia will be found which were clearly of embolic origin. Of these, 8 were on the right side of the body, and 5 on the left; the mitral valves alone were the seat of disease in 7 instances; both the mitral and the aortic valves, in 4; and the aortic valves alone, in 2 cases. I have met with only one example (No. 13, Table XII.) of sudden loss of vision from thrombosis, with retinal hæmorrhage. It would thus appear that the liability to embolic hemiplegia is much greater in connexion with mitral than with aortic valve disease. I may add, that this liability attains its maximum in cases of mitral stenosis.

Plugging of the peripheral branches of the internal carotid artery, by migratory clots detached from the valves of the heart, is readily explained by reference to the direct route of transit which that vessel supplies for these bodies. Hence, the frequent implication of the brain and the eye. It is, however, remarkable, and at present inexplicable, that the left hemisphere of the cerebrum, and the left eye, should be more frequently the seat of these lodgments than the right; yet so it is. In my 13 cases, the proportion of left cerebral thrombosis to right was as 8 : 5.

The middle cerebral artery, being the largest and the most direct of the terminal branches of the internal carotid, is most frequently the seat of the plug; next, in the order of frequency, are the anterior cerebral, and the ophthalmic.

The mechanical result of plugging in the cerebral or retinal arteries is similar to that exhibited in the lungs from embolism of the branches of the pulmonary artery (p. 910); namely, vascular engorgement and extravasation on the distal side of the clot.‡

* *American Journal of Medical Science*, January, 1874.

† Case 7, *loco citat.*

‡ See Case 111, p. 942

The gravity of the effects of this local disorganization must be necessarily proportionate to, (a) the delicacy of structure, (b) the importance of function, and (c) the inadequacy of the collateral circulation of the part. In the portion of the brain usually affected by an embolus, namely, that supplied with blood by the middle cerebral artery, and in the retina, these three conditions represent a maximum quantity. Hence, suspension and subsequently loss of the function of the corpus striatum and the adjacent portion of the cerebrum, from blocking of the middle cerebral artery; hence, likewise, similar results in the eye, from embolism of the central artery of the retina.

Professor Trousseau believed that mere occlusion of the middle cerebral artery will not afford an adequate explanation of the results which are apparently connected with it, and that the pressure of the clot upon the cerebral substance must be regarded as an important factor.* But the position and the minuteness of the clot render the latter view inadmissible, whilst the absence of a collateral circulation in the portion of brain substance supplied by the middle cerebral artery, as correctly pointed out by the late Dr. Kirkes, renders the changes consecutive to closure of this vessel quite intelligible. Complete restoration of structure and of function may take place by early disintegration of the plug, but experience shows that such a result is exceptional.† I have witnessed partial, but never complete, restoration of motion and of vision in cases of embolic hemiplegia and amaurosis. It is worthy of remark, that the paralysis resulting from cerebral embolism affects motion only; sensibility is unimpaired, and the paralyzed limbs are, in many cases, even hyperæsthetic.

Capillary embolism, or *infarctus*, due to impaction of pulverized fibrin in the extreme vessels of a part, is usually attended with less grave consequences than embolism of the larger vessels, owing to the very limited district of tissue which it affects. It is most probable that the occurrence of transient vertigo and loss of vision, with inability to maintain the erect posture, or even momentary loss of consciousness, so frequently witnessed in connexion with valvular disease on the left side of the heart, is due

* *Opus citat.*, vol. iii., p. 415.

† See Case 88, p. 868.

to capillary embolism of the brain by the detachment of detritus from the diseased valves.* Hemiplegia, of greater or less duration, accompanied by impairment or loss of speech and of memory when the lesion is located in the left side of the brain, is occasionally the result of infarction of the cerebral capillaries; Case 100 (p. 887) affords a probable example of this kind.

Embolism of the pulmonary artery and its branches has been already discussed in regard to its pathology (p. 910).† The migratory clot is derived, most frequently, from the right side of the heart, and is consecutive to cardiac thrombosis; it is occasionally the result of detachment of a clot from an inflamed vein, as previously shown (p. 1023). The gravity of the symptoms of pulmonary embolism will depend upon: (a) the order of the vessel engaged; (b) the size of the plug; and, when the vessels of one side only are affected, (c) the actual state of the opposite lung at the moment of the accident. When it arises from a thrombus formed within the heart, the consecutive changes in the lungs are usually localized, and of limited extent; and the symptoms are those of pulmonary apoplexy, including hæmoptysis. Even in such cases, however, sudden death may occur from infarction of the trunk of the pulmonary artery and its branches in both lungs. Cases 124 and 125 afford examples of this kind. The previous disease, in such cases, is usually constitutional, not simply valvular; most frequently it is leucocythæmia.

When the trunk of the pulmonary artery is plugged, the impacted body is usually solid, and has been moulded in one of the larger veins, whence it has been conveyed by the blood-stream to the right side of the heart, and into the pulmonary artery. It may consist, however, in the extension of a thrombus from the right ventricle; but, in such case, it is of more recent formation, less firm, and rarely fills the vessel. Coagulation from stasis soon takes place in the branches of the vessel, and the original clot, thus extended by thrombosis, assumes a branched appearance corresponding to the ramifications of the pulmonary artery.

When the main trunk of the vessel is completely blocked,

* See cases of aortic and mitral valve disease, *passim*.

† *Vide* an exhaustive paper on this subject, by Dr. G. Yeo in *The Dublin Journal of Medical Science*, for March, 1875.

death is very rapid; indeed, almost instantaneous. The patient is seized with a feeling of suffocation, is greatly agitated, and cries out for air; the chest heaves, and air freely enters the lungs, but the blood is not aerated; the right side of the heart is surcharged and paralysed by over distention, whilst the left side is empty and spasmodically contracted. Hence, death is caused partly by asphyxia and partly by syncope. The veins are turgid, the surface of the body is livid, and bloody foam usually issues from the mouth of the corpse.

Doctor Ormerod has recorded the case of a girl, aged twenty, paraplegic, and long bedridden, who complained of a queer feeling in her throat; cried out, at intervals, "Oh! my breath," threw her arms about wildly, and died within twenty minutes from the time she first complained of illness.*

When the pulmonary artery is but partially occluded by a clot, the symptoms are less urgent, and less rapidly fatal; they closely resemble those of the collapse of cholera, as remarked by Dr. A. Carpenter.† Treatment can be of little avail in these cases. In examples of the more urgent form of the affection, no time is allowed for treatment of any kind; whilst in cases less rapidly fatal, the administration of alkalies, as previously mentioned (p. 1022), affords the only chance of alleviation. Venesection may assist in relieving distention of the right chambers of the heart, but it should be practised with caution, lest it favour coagulation by further enfeebling the heart. The two following cases of sudden death by pulmonary embolism have come under my notice.

CASE CXXIV.—History of Great Privation; Anæmia, Stupor, and Debility; Swelling of the Tongue and Gums, without Salivation or Fætor; Leucocythæmia; Temporary Improvement; Sudden Death. Autopsy: Enlargement of Spleen; Embolism of Pulmonary Artery, and General Thrombosis of its Branches in both Lungs.

George M., aged twenty-two years, was received into hospital, November 1st, 1865. He was a young man of some education,

* *London Medical Gazette*, vol. ix., p. 788.

† *Lancet*, September 23rd, 1871.

who, owing to family reverses, had passed through the several social grades of apothecary's assistant, seaman on board a whaler, and fisherman on board a smack. He had left the latter employment three weeks previously, and, owing to some cause which he declined to mention, was forced to seek shelter in an out-house at Howth, where, during the interval of three weeks, he lay on a heap of straw, almost without food or drink.

When admitted, he was remarkably pallid, with a sallow tint, very drowsy, but easily roused, and then quite collected, answering questions clearly. The body was plump, but soft and flabby; surface and extremities cold; pulse weak, regular, and 90 in the minute. The stomach was extremely irritable, retaining neither food nor medicine, the vomited matter being of a bilious character. Despite the most careful examination, I failed to detect any organic disease whatever. The treatment consisted in sedatives, wine in moderate quantity, and liquid food.

A few days before his death, the tongue, which had been previously pale and moist, became so swollen and so tender, that he could not protrude it, but there was no salivation or fœtor of breath, and he had not, as he averred, taken a particle of mercury. A drop of blood taken from a puncture in his finger, and examined microscopically, seemed like water stained with blood, the margin of the field of vision being perfectly limpid and uncoloured; the white corpuscles were greatly in excess of the normal proportion, and nearly equal to the red in number.

His condition seemed to improve, sickness of stomach had ceased, he took more food, and slept better.

On the 17th of November, after returning to bed from the water-closet, he was observed to become suddenly faint, and he died in the course of a few minutes.

The body was sent for dissection to the School of Medicine, Cecilia-street, and was there examined. There was a good deal of subcutaneous fat. The organs were all sound, with the exception of the spleen, which was three times the normal size. A cylinder of nearly uncoloured fibrin had passed from the right ventricle into the pulmonary artery, which it entirely filled; the branches of this vessel, throughout both lungs, were likewise occupied, but not completely filled, by firm clot of a darker colour,

which, on slitting up the vessels, was removed entire, and found to present an arborescent form. The effort involved in the visit to the water-closet was obviously the cause of dislodgment of the clot from the right ventricle; but of its previous existence, no evidence was afforded by symptoms.

CASE CXXV.—*Emphysema of the Lungs with Bronchitis; Pallor; Musical Note of Systolic Rhythm at Lower Sternum; Sudden Death by Syncope. Autopsy: Heart Enlarged and Fatty; Right Chambers Thrombosed, and Tricuspid Valve Looped up by a Band of Fibrin; Pulmonary Artery and its Primary and Secondary Branches Plugged; Congestion of Bases of Lungs; Slight Thickening of Roots of Aortic Valves.*

Thomas H., aged seventy years, a labourer, was admitted into hospital, March 27th, 1871. For many years previous he had been in delicate health, subject to cough and dyspnoea.

At the date of admittance, he was pale and sallow; breathing somewhat embarrassed; pulse soft, regular, and 96 in the minute; cough, with frothy mucous expectoration. Physical examination elicited evidence of pulmonary emphysema, with bronchitis. Over the lower part of the sternum, a musical note was occasionally heard to accompany the first sound of the heart. A blister was applied to the chest, a stimulant expectorant was prescribed, and a few ounces of whiskey were allowed daily.

On the following morning (28th) he made a good breakfast, seemed in his usual health, spoke to the patients who occupied the adjacent beds, and did not complain of any special feeling of illness. Within ten minutes of the time when he thus addressed the other patients, he was heard to give a slight moan, and a few minutes afterwards, was found by the Sister on duty in the ward, lying across the bed, and all but lifeless; in the course of a few minutes more he died without a struggle.

The resident pupil on duty, the late Mr. Curran, who witnessed his death, reported to me, that during the last moments of his life the man was pale, and that death occurred apparently by syncope.

On examination of the body, the lungs were found to be em-

physematous ; the bronchial glands were enlarged, of dark colour, and cartilaginous density. The bases of the lungs were congested, and the bronchial membrane throughout was of a morone tint. The primary and secondary branches of the pulmonary artery were nearly filled with dark brittle coagulum.

The heart was somewhat enlarged, fatty on the surface, and exhibited a large "milk spot" on the anterior surface of the right ventricle. A mass of nearly decolorized fibrin, of the size and figure of a filbert, occupied the right auricular appendix, whence a thick band extended into the ventricle, looping up, in its passage, the anterior segment of the tricuspid valve, and establishing a connexion with a net-work of dense yellow fibrin which was entangled amongst the fleshy columns of the right ventricle. A large dark clot occupied the pulmonary artery, and extended, as above stated, into its primary and secondary branches.

The left ventricle was thick and somewhat dilated, and contained a mass of dark clotted blood. Both the mitral and the aortic valves were competent, but the roots of the latter, at their junction, were hard and spiculated in two situations.

The structure of the heart was in the granular stage of fatty degeneration. The existence of a musical note, accompanying the first sound at the lower part of the sternum on the 27th, may be accepted as showing that, even then, coagula had been already formed on the right side of the heart. The clot in the pulmonary artery was of a darker colour than that in the ventricle ; nevertheless, it is more than probable that it had been formed in the ventricle, whence it was suddenly dislodged, and impacted in the pulmonary artery. This hypothesis can alone supply a rational explanation of the sudden death. Dark coagula are not unusual in connexion with retiform thrombosis in the right chambers of the heart ; they are of more recent formation, less dense, and more loosely connected with the walls of the ventricle ; hence they are more readily detached.

CHAPTER IX.

NEUROSES OF THE HEART.

NEUROSIS, a term by which is implied derangement of innervation without structural disease of the heart, may consist in excessive or defective nervous activity of the organ. To the former category belong hysterical, dyspeptic, and gouty palpitation; and to the latter, palpitation from anæmia, chlorosis, and nervous exhaustion, cardiac asthma, angina pectoris, and exophthalmic goitre.* Hysterical palpitation requires no special notice; it is characterized by the usual hysterical accompaniments. Palpitation arising from latent gout, or dependent upon the gouty diathesis, is characterized by flatulence, acidity of stomach, and the presence of lithates in the urine; it is amenable to remedies usually successful in the treatment of developed gout.

Case 126 (p. 1037) affords a good example of this form of the affection, and shows the benefit derivable from the treatment just indicated. The palpitation of anæmia and of chlorosis is readily distinguished by its associations, by which, likewise, an appropriate line of treatment is indicated. The derangement of the heart's rhythm, due to nervous exhaustion, and implied in the designation "irritable heart,"† includes not only palpitation from trivial excitement, physical or moral, but likewise sudden and extreme variations in the rate of the heart's action from purely emotional causes. Onanism, and the excessive use of tea and tobacco, are the ordinary causes of rhythmical derangement of this kind. Da Costa, however, has witnessed it, in numerous instances, in the persons of soldiers long exposed to anxious and fatiguing duty in the field during the late American war. In all such cases the left ventricle is in some degree dilated, from the twofold cause of

* Palpitation has been already referred to at p. 161.

† *Toner Lectures* (No. 3), by J. M. Da Costa, Washington, 1874; and *Irish Hospital Gazette*, December 15th, 1874.

malnutrition and over-action, and, not unfrequently, a systolic murmur of adynamic origin exists at the apex.*

The following example of neurosis, from the frequent use of strong tea, has come under my notice. Two brothers, engaged in the tea trade, whose duty as "tea-tasters" required them every day to sip strong infusions of different samples of the article, became pallid and nervous; the pulse became rapid under the most trivial excitement; the heart beat tumultuously; there was dizziness, and occasional inability to maintain the erect posture without support, accompanied by a tendency to fall backwards when the head was raised. Under a short course of treatment with quinine and strychnine, whilst giving up the use of tea at their meals, and carefully avoiding to swallow any portion whilst tasting it in their daily avocations, these gentlemen were quickly relieved from the symptoms above described.

The discontinuance of the habit of onanism, the use of bromide of potassium thrice daily, in doses of grs. xv, and cold showers, rarely fail to arrest the palpitation from sexual abuse, and if the disease be not inveterate, to effect a rapid cure. Iron and quinine are sometimes required in the after-treatment of such cases; these agents are especially indicated where an adynamic murmur exists at the apex of the heart.

I recently witnessed, in the person of a young man, aged nineteen, a remarkable example of functional derangement of the heart from nervous exhaustion and excitement combined. He had been reading for a competitive examination, and had, within a few hours previously, received the intelligence of his having obtained the first place. When he called on me for the purpose of obtaining a certificate of health for the Civil Service, he was returning home from the school where he had been educated, after announcing his success, and receiving an ovation from his fellow-students. He was pale and fidgety; the pulse was 150, and intermittent; the impulse of the heart was strong and abrupt, and accompanied at the apex by a very distinct thrill, whilst the sounds were sharp and loud. I directed him to return in the course of the day, after he had taken a walk and some refresh-

* See pp. 232 and 568.

ment. On his second visit, two hours later, the pulse was 80 and free from intermissions; the action and sounds of the heart were normal, and fremitus no longer existed.

Professor Laycock thinks that extreme nervous excitability of the heart, such as that exhibited in the preceding case, is premonitory of organic disease;* I cannot agree with him in this opinion.

The subjoined tracing, though not exhibiting irregularity, shows the character of the pulse in some cases of irritable heart.

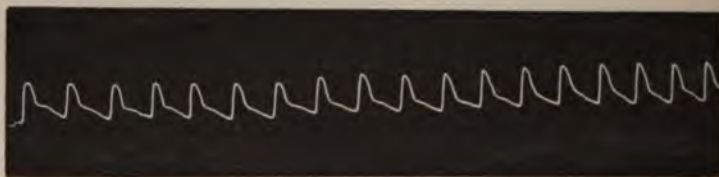


FIG. LXVII

Irritable heart, with anæmia (Meehan).

Tracing taken June, 1873.

Cardiac asthma is a peculiar form of dyspnœa, of an intermittent character, and dependent upon organic, and most frequently, valvular disease of the heart. It is characterized by restlessness and agitation, craving for air, quick and deep respiration, congestion of the face, palpitation, and increased rapidity of pulse. In those who are subject to it, the paroxysm is readily induced by physical or mental excitement. It depends upon temporary derangement of balance between the circulatory and respiratory functions, consequent upon instability of cardiac innervation. I have most frequently witnessed it in connexion with disease of the aortic valves and aorta.†

In the following case, however, there was no valvular disease, whilst undeveloped gout was obviously the cause of the paroxysmal seizures.

* *Dublin Journal of Medical Science*, July, 1873.

† See Case 80, p. 858; Case 87, p. 866; Case 101, p. 889; Case 102, p. 918; and Case 106, p. 927.

CASE CXXVI.—*Paroxysms of Dyspnœa, with Palpitation and Flatulence, in Connexion with Undeveloped Gout. Recovery.*

Mr. R., aged fifty years, superintendent of a large mercantile establishment, consulted me on the 12th of January, 1870. Three years previously he had experienced his first symptoms, in Paris. He was anxious looking, haggard, and somewhat excited by his visit to me. Pulse 102, and weak but regular; apex-beat in nipple-line; sounds, especially the first, rather clear, but unaccompanied by thrill or murmur. Family gouty; has been himself repeatedly threatened with gout on taking malt drinks; urine deposits a lateritious sediment. He is occasionally, towards morning, suddenly awakened from sleep by a feeling of great oppression of the chest, with palpitation, which obliges him to start up, raise his arms, and struggle for breath. The paroxysm lasts about five minutes, after which he has complete relief. Has been taking brandy rather freely to relieve these symptoms. Moderate exercise agrees with him; he is subject to flatulence, and is relieved by eructation; bowels regular. To have light nutritious diet, and a moderate allowance of claret, but no brandy; and at night, a draught containing ℥xx of liquor of the hydrochlorate of morphia, with ℥ij of hydrocyanic acid, and ℥x of chlorodyne, in an ounce of water.

On the night of the 16th he had the premonitory sensation of a fit, which he managed to ward off by "shaking himself up." This feeling recurred five times in succession on going to sleep, and on each occasion he succeeded in averting a fit by the manœuvre just described. He ultimately fell asleep after taking some brandy and solid food.

17th. Pulse full, regular, 96; urine clouded, and acid. To have a belladonna plaster to precordium, and every morning a pill composed of aromatic powder and mercurial pill, of each gr. ij, with gr. j of the acid extract of colchicum. A draught, consisting of tincture of Indian hemp, ℥xv, hydrocyanic acid, ℥ij, and chloric ether, ℥x in an ounce of water, to be taken at night, if necessary; and Vichy water (L'Hôpital) in the course of the day.

21st. Pills act twice daily; urine now clear; has had only

one slight paroxysm since last report. To continue pills and Vichy water.

February 2nd. Much improved. No recurrence of dyspnoea or palpitation. Pulse 72, regular. Urine clear. Slight muscular pains in thighs and upper arms; but, with this exception, he regards himself as well.

The group of symptoms implied by the designation *angina pectoris* has been long familiar to clinical observers, at least in general outline. Typical examples may be found recorded by Morgagni.* Heberden, however, was the first who employed the name "*angina pectoris*," and gave a special individuality to the symptoms included under it, by presenting them in a collective form, and connecting them, theoretically, with a particular morbid condition which he designated "spasm" (distention).† He does not indicate precisely the seat of assumed "spasm," which, a few years later, was localized in the heart by Macbride.‡ Shortly after the publication of Heberden's memoir, an anonymous correspondent, who identified the disease in his own person, by means of the description given of the symptoms by Heberden, presented him with a graphic sketch of his own case, dwelling especially upon the substernal pain, shooting across the chest and down the arms as far as the elbows, and the accompanying dyspnoea, during the paroxysm. He bequeathed his body to Heberden for examination after his death, which took place in one of these attacks; the body was examined by John Hunter. The only lesion discovered was slight ossification at the root of the aorta.

Dr. Fothergill's paper was, after Heberden's, the next contribution on the subject.§

Doctor Wall, of Worcester, communicated to Heberden the particulars of a case of *angina pectoris* which had been under his observation. After death, the heart was found to be fatty,

* *De Sedibus et Causis Morborum*, liber ii.

† *Medical Transactions*, No. VI., vol. ii., July, 1768. See also *Commentaries on the History and Cause of Diseases*, 1802.

‡ *Medical Observations and Inquiries*, vol. vi., 1778.

§ *Ibid.*, vol. v., 1773.

the aorta was dilated and atheromatous, and the aortic valves were ossified.*

Doctor Butter published a special work on this subject,† in which he endeavoured to show that angina pectoris is a manifestation of gout, often ceasing entirely with a fit of that disease.

Eight years later, Dr. Parry's well known treatise appeared,‡ and contained the best exposition of the subject presented up to that date. Dr. Parry, having reference to the two most prominent symptoms of the affection, namely, faintness, and painful constriction of the chest, named it *syncope anginosa*. He adopted the view first propounded by the celebrated Edward Jenner, of the dependence of angina pectoris upon ossification of the coronary arteries, regarding calcareous change with dilatation of the aorta, and enlargement of the heart, as subordinate causes, and attributing the attendant pain to accumulation of blood in the heart and large vessels. He admitted that gouty patients are the most liable to attack, but considered that organic disease of the heart, so commonly associated with chronic gout, was the cause of this proclivity.

It is unnecessary to enumerate the several memoirs which have been since written to illustrate the subject of angina pectoris. Many of these will be necessarily referred to in the sequel.

M. Jaccoud§ divides angina pectoris into true and false, after Lartigue. The true form, according to him, may be idiopathic or primary, or it may be symptomatic. It may be doubted, however, that angina pectoris is ever strictly primary, except in the few instances in which it is the only actual indication of gout. Trousseau, indeed, held that in many cases it is nothing else than "partial epilepsy," or "epileptic neuralgia."|| But few, I apprehend, will adopt this view. He admitted, also, that it may arise from gout or rheumatism.

In the great majority of examples of angina pectoris, organic

* *Medical Transactions*, vol. iii., 1785.

† *A Treatise on the Disease commonly called Angina Pectoris*, 1791.

‡ *An Inquiry into the Symptoms and Causes of the Syncope Anginosa*, London, 1799.

§ *Dictionnaire de Médecine et de Chirurgie*, vol. ii.

|| *Clinical Medicine*, Sydenham Society's Edition, vol. i., p. 65.

disease of the heart is clearly revealed by physical evidence, and in a still larger percentage of cases, *post mortem* examination would demonstrate its presence. Valvular disease, which, in cases of angina pectoris, is most frequently aortic, and likewise dilatation and atheromatous change of the aorta above the valves, may be detected by physical examination. Not so, however, in some instances, fatty degeneration of the heart; but the symptoms indicative of this condition are usually so well pronounced in the exceptional cases, as to supplement the want of physical signs, and leave very little doubt as to the existence of fatty change of the heart.

Aneurism of the aorta has been occasionally met with in connexion with angina pectoris; but, as most cases of aneurism have not been associated with angina pectoris, and as the latter is by no means a characteristic sign of the former disease, the association must be regarded as a coincidence. Calcification of the aorta and of the coronary arteries, and fatty degeneration of the heart, so frequently witnessed in connexion both with aneurism and angina pectoris, may serve to explain the coincidence. It may be useful here, as exhibiting the unsettled state of opinion with reference to the pathogeny of angina pectoris, to summarize the views held as to its cause.

- (a) Spasm (distention); Heberden.
- (b) Spasm of the heart; Macbride, Latham.
- (c) Calcification of the coronary arteries; Jenner, Parry.
- (d) Spasm of the heart, and pressure of the cardiac nerves against calcified coronary arteries; Sir Everard Home.
- (e) Neuralgia located in the pneumogastric, sympathetic, and brachial nerves; Desportes, Laennec, Piorry, Flint, Jacoud.
- (f) Nervous and spasmodic affection arising from gout; Butter, MacQueen, Johnston, and Blackall.
- (g) Hyperæsthesia of the cardiac plexus; Romberg.*
- (h) Hypercinesia, with hyperæsthesia; Bamberger.†

It is, I think, essentially a neurosis; engaging primarily, in most instances, the cardiac plexus, whence it is reflected through

* On the authority of Niemeyer.

† *Ibid.*

the sympathetic to the spinal nerves, and occasionally through the pneumogastric to the cesophageal plexus. It is, in short, a cardialgia of the most aggravated character, which may or may not be associated with organic disease of the heart. But in the great majority of cases, it is so associated. Of 19 cases of angina pectoris collected from the *Transactions of the Pathological Society of London*, by Dr. J. W. Ogle,* the coronary arteries were calcified in 12; the heart was fatty in 12; the aorta was atheromatous in 10, and valvular disease existed in 6 cases. Grave structural changes of the aortic and cardiac plexuses have been found by Lancereaux.†

The occurrence of death during a paroxysm must be attributed to syncope from excessive pain in a vital organ, weakened by disease. Out of 13 cases of angina pectoris reported by Dr. Ogle, in which *post mortem*‡ examination showed the existence of atheroma of the aorta, with partial or complete occlusion of one or both coronary arteries by calcareous matter, or by fibrin, sudden death occurred in 11 instances, and in 3 of these the heart was in a state of fatty degeneration. Hence, I regard the designation "syncope anginosa,"§ as most appropriate.

I cannot look upon angina pectoris as necessarily fatal. In the primary form it certainly should not be so regarded; but the cases in which it is primary are comparatively few. The mortality of the disease is due rather to its usual associations, organic disease of the heart or aorta, than to its essential gravity. Flint has reported several cases in which, after repeated attacks, the patients were exempt from the affection for many years.|| I have met with a few cases of the same kind. Case 44, p. 647, and Case 93, p. 876, may be referred to as examples.

The immediate cause of the paroxysms is, most frequently, quick walking soon after meals, the ascent of an acclivity, movement against the wind, or emotional excitement, especially

* *British and Foreign Medico-Chirurgical Review*, No. 92, October, 1870, p. 449.

† *Gazette Médicale*, 1867, p. 432.

‡ *Loco citat.*

§ Parry.

|| *Diseases of the Heart*, second edition, 1870.

anger. The patient then suddenly feels an excruciating pain of a constrictive character shooting across the chest at the level of the middle or lower portion of the sternum, and extending down one or both arms, most frequently the left; it not unfrequently radiates to the shoulders and back of the neck, round the chest in the course of the thoracic and intercostal nerves, and over the abdomen and groins, following the course of the branches of the lumbar plexus. The sufferer is forced to stand still and seek support, has a feeling of impending death, looks pale, haggard, and greatly alarmed, and fears to breathe lest he aggravate his pain. The pulse usually maintains its normal rate and volume, but exhibits occasional intermissions; it may, however, be very slow or very rapid during the paroxysm. There is no palpitation, and air freely enters the lungs when the patient essays to breathe. After a period of a few seconds, or a few minutes, the paroxysm ends quite as suddenly as it commenced, and generally with eructation of flatus. The patient is then in his normal condition, but is puzzled as to the nature of his attack, and alarmed at the prospect of its recurrence. I have known spasm of the cesophagus, as indicated by globus, to characterize the earlier invasions of the disease. Sighing is usually a concomitant symptom, whilst the posture assumed by the patient may vary from the erect to the bent. The intervals between these seizures are of uncertain length, and depend, in a great degree, upon the habits and temperament of the patient. At first they are usually considerable, varying from days to months, or even years. I have known a gentleman to suffer only one attack, now many years ago. He is at present in excellent health; but in his case gout, of which he subsequently had repeated fits, was the only assignable cause, and his immunity since that date is due to his active habits and caution as to food and drink.*

Unfortunately, however, the fits usually recur with progressively increasing frequency; they are likewise more severe and protracted in the progress of the case; and towards its termination several may occur within the twenty-four hours, being provoked by the slightest movement or chill, even that involved in getting into or out of bed.

* See Case 128.

The disease may be fatal within a fortnight after the first seizure, or even in the first attack.* The celebrated Dr. Arnold, of Rugby, died in less than three hours from a primary invasion of angina pectoris.†

Genuine angina pectoris may be distinguished from the less grave affections, cardiac and spasmodic asthma, by the presence of the characteristic substernal pain, and by the absence of quick, laboured, and sonorous respiration, craving for air, agitation of the body, palpitation, and rapidity of pulse. In the cardiac form, asthma is further characterized by engorgement of the cervical veins, congestion of the face, and by the signs of distention of the right side of the heart. Laennec obviously failed to draw this distinction, hence the less grave aspect which angina pectoris presented to him.

According to Pinel, angina pectoris is more common in winter than in summer;‡ a difference which is probably due to the unfavourable effect of cold upon the surface of the body. It would seem that England enjoys an unenviable distinction above all other countries by the prevalence of the disease amongst its population. The rich are certainly more liable than the poor, and preeminently those of high intellect and anxious temperament. The male is more frequently affected than the female sex. Of 88 cases collected by Sir John Forbes, only 8 occurred amongst females.§ The age of the persons attacked is rarely under forty-five.

Angina pectoris is a comparatively rare affection. I have met with only 6 genuine examples. In 3 of these the disease was connected with latent gout, in 2 with fatty disease of the heart, and there was no organic lesion in one instance. As to the association of angina pectoris with valvular disease, I will only remark, that out of a total of 136 cases of valvular lesion recorded in my notes, angina pectoris was present in 3 only.

Of 188 cases of organic disease of the heart, noted from personal observation by Flint, only 8 exhibited angina pectoris.

* Latham on the *Diseases of the Heart*, vol. ii., Lectures xxxvii., xxxviii.

† See his life, by Dean Stanley.

‡ Jaccoud, *loco citat.*

§ *Cyclopædia of Practical Medicine.*

The prognosis is eminently grave, even in the most favourable cases, owing to the possibility of latent organic disease of the heart or aorta; but where undeveloped gout is, presumably, the only cause in operation, a less unfavourable prognosis may be given. Where organic disease of the heart or the aorta manifestly exists, no hope of recovery can be entertained, although life may, even in such cases, be prolonged for years.

The treatment of the disease resolves itself into that which is proper during the paroxysm, and that which should be pursued in the intervals with a view to averting its recurrence.

The treatment of a patient in a paroxysm of angina pectoris should be regulated by the most urgent symptoms present. If, in any degree, venous turgescence were exhibited, I would not hesitate to draw blood by venesection, or by cupping at the precordium, to the amount of four or five ounces. I should expect that a weak and intermittent pulse, associated with superficial congestion, would expand and become steady under partial abstraction of blood, owing to the relief thereby given to the distended right ventricle. Both Trousseau and Jaccoud object to blood-letting in this disease under any circumstances, but, as I believe, on insufficient grounds. Where pallor without congestion characterizes the superficial surface, as is usually the case, dry-cupping over the heart, followed by a sinapism or a stimulant embrocation, would be more appropriate. If pain were the prominent symptom, opium with ether should be given at the same time, 3ss to 3j of Hoffinan's anodyne with ℥xxx of the liquid extract of opium. Sulphuric ether, given by inhalation, to the amount of ʒiij to ʒiv, might be substituted for the preceding, or, better still, the nitrite of amyl, from ℥v to ℥x, which, in many cases, promptly arrests the paroxysm.* The inhalation of nitrite of amyl is not, however, free from danger, and should be administered with great caution, especially where organic disease of the heart or aorta is known to exist. Dr. Haddon mentions two cases in which the most alarming symptoms,

* *Vide* a most interesting report of his own case by Dr. Herries Madden, *Practitioner*, December, 1872; see also Lauder Brunton, *Lancet*, July 27th, 1867; Leishman, *Practitioner*, September, 1869; Wilks, *Lancet*, January 16th, 1869; H. C. Wood, *American Journal of the Medical Sciences*, October, 1871; and Haddon, *Edinburgh Medical Journal*, July, 1870.

including stoppage of the heart's action and general congestion, followed its use. In view, however, of the not improbable existence of fatty disease of the heart, it would be in the highest degree imprudent to venture upon the administration of chloroform. I cannot agree in the opinion expressed by Niemeyer, that opiates and other narcotics are to be avoided.* On the contrary, the hypodermic injection of morphia, or of atropia, is often followed by great relief.

Where faintness is the principal symptom exhibited, brandy should be freely given till reaction has been induced. Warmth should be, at the same time, applied to the feet, and the best form in which to apply it is that of a warm mustard foot bath. In the last mentioned cases, viz., where great debility is the chief characteristic of the paroxysm, Duchenne and Aran have successfully practised electrization of the left side of the thorax. Both these physicians have reported examples of apparently complete cure effected by electricity so applied.† Eulenburg, of Berlin, has likewise had some success with electricity; he prefers the constant current (up to 30 elements of Siemen's), the positive pole being applied to the sternum by means of a broad plate, and the negative in the situation of the sympathetic chains in the neck. The cases best adapted for the use of electricity are, he thinks, those which exhibit irritation of the excitomotor or the vaso-motor centre, as shown by small and hard pulse, and more or less excitement of the heart.‡

In the treatment of the patient during the interval between the fits, the physician should be guided by the conclusion at which he may have arrived, as to the cause of the affection in the case before him. If it were manifestly latent gout, the appropriate remedies in the treatment of that disease, and preeminently the salts of lithia, with a suitable diet and regimen, should be prescribed, whether organic disease existed or not. Case 128, p. 1049, affords a good example of the beneficial action of lithia and regulated regimen in the treatment of angina pectoris of gouty origin. Chloral hydrate given every

* *Text-Book of Practical Medicine*, vol. i., p. 371.

† See Trousseau, *Clinical Medicine*, vol. i.

‡ *Medical Times and Gazette*, May 7th, 1870.

night, in doses of grs. xxv to xxx, in combination with sulphuric ether and aromatic spirit of ammonia, has been found by Dr. Strange to give great relief. After the continuous use of the drug for four months, an additional half dose, every night, became necessary.* Fothergill has mentioned a case in which, under the use of the Bath waters, an apparently perfect cure was effected, the patient having had no fit for a period of twenty years.† Trousseau recommends Fowler's solution, and also the simultaneous use of bicarbonate of soda and belladonna, administered according to the method of Bretonneau; viz., in doses gradually and alternately increasing and diminishing at intervals, for a protracted period.‡ Anstie also strongly recommends Fowler's solution, in doses of ℥v, thrice daily; and where the stomach will not tolerate this, he suggests the sub-cutaneous injection of strychnia, to the amount of the one-hundred and twentieth to the one-hundred and sixtieth of a grain.§

With a similar object in view, namely, that of arresting degeneration of tissue by promoting general nutrition, phosphorus may be administered in minute doses, one-fortieth to one-sixtieth of a grain twice daily, as recommended by Dr. Broadbent.|| I have no personal experience of this agent in the treatment of angina pectoris; but, encouraged by the results of Dr. Broadbent's experience, I would certainly give it a trial in suitable cases.¶

Doctor Rufus K. Hinton recommends the use of bromide of ammonium as a prophylactic in this disease. He publishes two cases of indubitable angina pectoris, in which, given in doses of grs. xv to xx, the bromide of ammonium would seem to have been efficacious in preventing the paroxysms.*

In addition to the examples of angina pectoris incidentally

* *Medical Times and Gazette*, September 4th, 1870.

† *Loco citat.*

‡ Trousseau, *opere citat.*

§ *British Medical Journal*, November 11th, 1871.

|| *Practitioner*, January, 1875.

¶ For a full *résumé* of the subject of angina pectoris, especially in reference to treatment, see *The Boston Medical and Surgical Journal* of May 21st, 1874.

** *Philadelphia Medical and Surgical Reporter*, March 6th, 1875.

recorded in the preceding pages (Case 44, p. 647; Case 47, p. 681; and Case 101, p. 889), the two following cases may be given here.

CASE CXXVII.—*Angina Pectoris of the Ingravescens Character; Double Aortic Murmur; Improvement under the Use of Lithia and Chloral Hydrate; Intercurrent Jaundice; Death by Exhaustion. Diagnosis: Hypertrophy of the Left Ventricle; Obstruction and Inadequacy of the Aortic Valves, and Dilatation and Atheroma of the Aorta.*

Mr. S., aged forty-two years, a hard-working barrister, of anxious temperament and regular habits, consulted me on the 2nd of July, 1873. Had not had rheumatism or gout. Two years previously, he experienced, for the first time, pain across the front of his chest, and lasting about ten minutes, after exertion. He had another similar attack a few weeks afterwards. Latterly, these "spasms," as he termed them, had been more frequent. They occurred most frequently at night, but seldom in bed, and never, except after exertion of some kind. They had repeatedly occurred on getting out of bed in the morning. Exposure to cold, or the application of cold to the surface, such as washing with cold water, was certain to bring on a "spasm"; walking against the wind had a similar effect.

Some of these attacks had been very mild, consisting only of pain across the chest lasting for a few minutes, whilst others had been very severe, attended with excruciating pain in the chest, back of the neck, and down the left arm, to the back of the hand and fingers. These latter had been accompanied by strong and rapid pulsation of the heart and gasping for breath, obliging him to support himself against some fixed object. Food of certain kinds, such as pastry, which caused flatulence, rarely failed to bring on a fit.

He was remarkably anxious-looking and haggard; could sleep well, and usually on the left side. Pulse 84, full, soft, and regular, but collapsing, and visible after excitement. The apex of the heart pulsated feebly in the nipple-line; first sound at apex very faint; second sound replaced by a loud diastolic murmur, transmitted from the base. This latter murmur was audi-

ble over the entire front of the chest, but with greatest intensity at the lower portion of the sternum.

Over the base of the heart, a faint but rough systolic murmur was likewise heard; it was transmitted through the arch of the aorta, and into the carotids. The action of the bowels and kidneys was regular, but the urine occasionally exhibited a deposit of lithates. An opiate plaster was applied to the precordium; he was directed to apply a few leeches at midsternum, or to be dry-cupped in that situation, in the event of a paroxysm of pain occurring; also to take, thrice daily, ℥ij liquor. strych., (B. P.), with ℥ij tinct. digitalis, and grs. iss sulph. quiniæ, in solution.

On the 28th of October he was slightly jaundiced, and the liver was congested and tender; grs. vj of the citrate of lithia were prescribed daily, and likewise an aperient pill containing inspissated ox gall. A draught, containing grs. xx of chloral hydrate was directed to be taken at night should a paroxysm occur.

On the 26th of December, he reported himself as greatly improved. Since last visit he had been rarely attacked with pain in the chest or arm; could sleep through the night, and on two occasions when the "spasm" came on in bed, he was able to control it by means of the chloral draught. On the preceding day he had walked ten miles without an attack, or feeling fatigued. Had cough, with expectoration of viscid mucus, and suffered also from flatulence. For the former, ℥xxx of compound tincture of camphor, and of syrup of cinchona, with ℥vij of cherry-laurel water were prescribed, and, for the latter, Belloc's charcoal.

In March, 1874, the paroxysms became more frequent and more severe; the pain then radiated from the epigastrium to the back and over the abdomen; the jaundice returned, the appetite failed, and he could not obtain sleep. He wasted considerably. Cough was more urgent, and was attended with slight hæmoptysis. The face assumed an anxious and haggard expression; the feet became swollen; œdema of the lungs set in; he could rarely assume the recumbent posture, even for a moment, owing to extreme dyspnoea, and died, worn out, on the 13th of May. The body was not examined.

CASE CXXVIII.—*Latent Gout ; Angina Pectoris accompanied by Intermissions of the Action of the Heart ; Slow and Suspicious Breathing during Sleep ; Nephritic Colic ; Articular Gout ; Recovery under the Use of Lithia and Alkalies.*

A professional man, in good business and of active habits, aged about forty-five years, naturally florid, but latterly become fat and pale, consulted me in January, 1868, in reference to palpitation, accompanied by intermissions of the cardiac impulse and of the radial pulse, which he latterly began to experience on going up stairs, or being otherwise hurried in his movements ; he had also felt, on these occasions, a dull tensive pain behind the sternum. He further informed me that, during sleep, his breathing had been noticed, of late, to be occasionally slow, sighing, and oppressed. Pulse 86, and regular ; appetite indifferent ; has had flying gouty pains in the feet and in the muscles of the chest. Lithia was prescribed, and warm mustard foot baths were directed, with a view to develope an attack of gout in the feet.

I heard nothing further of this gentleman's health till February, 1870. He had been fairly well during the interim, and continued to discharge his professional duties, which were laborious, without interruption. He had, however, been much annoyed by acidity of stomach, and had noticed, from time to time, a deposit of lithates in the urine.

On the night preceding my visit to him he had a severe attack of nephritic colic, after having dined heartily upon salmon. He was quickly relieved, under active treatment by means of counter-irritants and alkaline diuretics, and, in the course of a couple of days, was able to resume his professional duties.

January, 1875. Since last report he has had repeated attacks of gout in the feet, and, for some time after each of these attacks, he has felt much better in regard to his general health. No return of angina, from which he has been now free for more than seven years. Habits most carefully regulated in regard to diet and exercise.

The complex morbid condition known under the designation of

Exophthalmic Goitre, Graves' disease, or Basedow's disease, which has been already referred to at p. 489, consists in its complete form, of the "symptomatic triad," palpitation with throbbing of the arteries of the neck, vascular turgescence and enlargement of the thyroid body, and prominence of the eyeballs, was first described by Graves in his clinical lectures delivered in 1835. Flajani, in 1802, had published three cases, now regarded as belonging to this category, amongst the symptoms of which, however, exophthalmia was not included.* Galezowski† claims for Demours the original description of the disease in 1818. Parry, in 1825, recorded seven cases of thyroid enlargement in connexion with affections of the heart.‡ According to Hirsch, Basedow was the first writer who described the disease; he proposes therefore to name it after him; Basedow's account of the affection was, however, not published till 1840.§ Jaccoud has shown|| that to Graves is due the merit of having been the first to direct attention to this singular affection as a distinct morbid entity.

According to Dr. Stokes, the essential features of the affection are the following:

1. Increased force and rapidity of the heart's action, of long continuance, and without fever.
2. Excited action of the carotid and thyroid arteries.
3. Enlargement of the thyroid gland, varying with the force of the heart.
4. Enlargement of the eyeballs, without any disease of the orbits or brain.

He regards it as "a special form of cardiac neurosis, which may result in organic disease," and, probably, a similar affection of the cervical vessels.¶

Niemeyer's view of the pathology of the disease is virtually

* *Collezione d'Osservazioni*, tom. iii., p. 270.

† *Gazette des Hôpitaux*, 1871, p. 425.

‡ Collections from the unpublished writings of the late Caleb H. Parry, M.D., London.

§ Casper's *Wochenschrift*, March, 1840.

|| *Leçons de Clinique Médicale de R. J. Graves, ouvrage traduit et annoté par le Docteur Jaccoud*, 1862, tom. ii., p. 290.

¶ *The Diseases of the Heart and Aorta*, p. 279.

identical with that of Stokes. He attributes the affection to palsy of the vaso-motor nerves, which, in his opinion, "fully accounts for the dilatation and increased pulsation of the carotid and thyroid arteries, as well as for the cedematous swelling of the thyroid gland and intra-orbital fat." There is likewise, he thinks, "a sub-paralytic state of the vessels of the muscles of the heart."*

Trousseau, while admitting that neurosis of the heart constitutes an essential element of the disease, does not regard it as the primary lesion. He looks upon the affection as "a neurosis of the sympathetic, from congestion or structural change of the ganglionic system." In one instance he found, on *post mortem* examination, the inferior cervical ganglia of the sympathetic, especially that of the right side, greatly enlarged, and highly vascular; its connective tissue hypertrophied, and exhibiting nuclei and fusiform cells, whilst the proper nerve constituents, both cellular and fibrous, were wasted. The cardiac plexus was simply vascular.† Drs. Cruise and R. M'Donnell found the inferior cervical ganglion almost entirely replaced by areolar and adipose tissue.‡ According to Dr. J. Begbie, the disease is essentially anæmic.§ Bellingham|| and Makenzie,¶ were of the same opinion, whilst Dr. J. W. Begbie regards anæmia as the primary condition, vaso-motor paresis being a direct consequence of this.**

Doctor Lauder Brunton is of opinion that the palpitation, so characteristic of this disease, is due to direct stimulation of the accelerating nerves of the heart, which descend from the vaso-motor centre in the medulla oblongata in company with the vertebral artery, and, after passing through the inferior cervical ganglion of the sympathetic, are supplied to the heart. Irritation of this ganglion would, therefore, account for cardiac excitement.

The remittent engorgement of the thyroid vessels may, in

* *Text-Book of Practical Medicine*, vol. i., p. 375.

† *Clinical Medicine, Sydenham Society's Edition*, vol. i., p. 575-82.

‡ See abstract of Case, p. 1061.

§ *Monthly Journal of Medical Science*, 1849, p. 495, and *Contributions to Practical Medicine*, p. 176.

|| *Diseases of the Heart*, p. 582.

¶ *Diseases of the Eye*, 1854.

** *Edinburgh Medical Journal*, vol. ix, part i., p. 206.

his opinion, depend upon paralysis of their vaso-motor nerves which are derived from the second cervical ganglion, or upon "inhibition" of these through other nerves. He thinks that protrusion of the eyeballs is most probably due to turgescence of the blood-vessels and lymphatics in the orbit; remarking that, in the experiments of Bernard, exophthalmia was readily produced by irritation of the nerve-filaments connecting the first and second dorsal ganglia of the sympathetic with the spinal cord.

Finally, he calls attention to the loss of *consensus* between the movements of the eyeball and those of the upper eyelid, which was exhibited in one of his cases, and ventures to account for this symptom on the assumption of disturbed innervation of the lids, as propounded by von Gräfe; and especially of the upper lid, through the levator muscle, which derives its nerve supply in part from the sympathetic.*

Mr. Swanzy has reported a case of exophthalmic goitre, in which the want of consensual movement of the upper eyelid, when the patient looked down, was well pronounced; nictitation was likewise remarkably slow and infrequent in this case, both phenomena depending, in his opinion, upon deranged innervation of the lids.†

Whilst I believe that the singular combination of symptoms constituting exophthalmic goitre is essentially dependent upon vaso-motor paresis, I feel with Dr. Cheadle that the frequent coexistence of dilatation of the pupils, a condition which indicates rather irritation than paralysis of the sympathetic nerves, with elevation of temperature, epistaxis, and diarrhoea, constitutes an objection which must be answered before this theory can be unreservedly adopted. Such an answer cannot at present be given.

Cardiac excitement is usually regarded as the primary symptom. Trousseau, however, recognizes a previous state of nervous irritability, manifested by peevishness and impatience. This is soon followed by a sense of fulness and throbbing in the head and eyes, palpitation, throbbing at the epigastrium, and often vomiting and diarrhoea. The patient now loses flesh, notwithstanding the existence of a morbid appetite, which, however, is

* *St. Bartholomew's Hospital Reports*, vol. x., 1874.

† *Irish Hospital Gazette*, vol. i., p. 258.

soon replaced by anorexia. In females, derangement of the menstrual function is an early symptom, and, as noted by Dr. Cheadle, constant elevation of temperature and occasional profuse sweating and epistaxis.* The other two cardinal symptoms now gradually declare themselves, enlargement of the thyroid usually preceding the exophthalmia. The order of the two latter symptoms may, however, be inverted, as it was in the majority of my cases; or either may be absent, most frequently the thyroid enlargement, constituting an example of the "incomplete" form of the disease.

Exophthalmic goître may be acute or chronic. Dr. Adams has known ocular prominence to be suddenly produced by a violent fit of coughing and retching,† and Dr. R. Taylor has witnessed its occurrence in the course of one night.‡ Trousseau has recorded the case of a female, sixty years of age, in whom the three prominent symptoms of the affection were developed in the course of a single night from excessive mental emotion.§ The disease is, however, most frequently of slow and progressive growth, the rate of its progress being unsteady and fitful. A fright or a mental shock of some kind is usually the starting point of the symptoms. Protracted watching and great anxiety may give rise to it. Stokes has known it to be produced in the male subject by long continued bleeding from hæmorrhoids.

The great majority of those who suffer from exophthalmic goître are females. Males are occasionally, though rarely, the subjects of it. I have not seen an example of it in the latter sex; but Stokes|| and Trousseau have each witnessed one example. Cheadle,¶ Moore,** Foot,†† Begbie,‡‡ and Reith§§ have each re-

* *St. George's Hospital Reports*, vol. vii., 1875, p. 81.

† Stokes on *Diseases of the Heart and Aorta*, p. 295.

‡ *Medical Times*, May, 1856, p. 516.

§ *Opus citat.*, p. 575.

|| For further report of this case by Dr. L. McDonnell, see *Dublin Journal of Medical Science*, vol. xxvii., p. 200.

¶ *St. George's Hospital Reports*, vol. iv.

** *Dublin Quarterly Journal of Medical Science*, November, 1865, p. 344.

†† *Irish Hospital Gazette*, vol. ii., p. 179.

‡‡ *Dublin Hospital Gazette*, vol. ii., p. 107, quoted from Case Book of *The Edinburgh Medical and Surgical Journal*.

§§ *Medical Times and Gazette*, November 11th, 1865.

corded a case of the disease in the male sex. Of 50 cases collected by Withusen, the subjects were males in only 8 instances.* The period of life at which it is most common is between puberty and the age of forty. From twenty to twenty-five years is, according to Trousseau, the age at which it usually occurs. Of my 6 cases, 3 were over twenty-five, and 2 were forty years of age; but, in the latter cases, the disease had been in existence six and eight years respectively when the patients came under my notice. Niemeyer remarks that men affected with this disease are usually advanced in life, whilst, in females, it generally appears in youth.†

Of the three characteristic symptoms of this disease, palpitation is usually the first exhibited. In Case 134, however, enlargement of the thyroid gland was the first symptom noticed; palpitation with further and rapid growth of the thyroid supervening six years later, on the occurrence of uterine hæmorrhage. The pulse is rarely under 120, whilst it not unfrequently attains to 160 in the minute. In cases uncomplicated by hypertrophy of the left ventricle it is small, and most frequently regular; but irregularity or intermittence of the pulse is occasionally witnessed in this, as in all other forms of cardiac neurosis.

The subjoined tracing, taken from a female suffering from exophthalmic goitre in a typical form, the notes of whose case I have unfortunately lost, shows well the irregularity and intermissions which characterize the pulse in many cases of the affection.

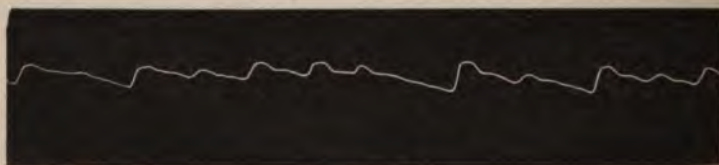


FIG. LXVIII

Exophthalmic Goitre. (McG.)

Pulse 70. September 17th, 1874.

The impulse of the heart is abrupt and energetic rather than

* *Dublin Medical Press*, July 6th, 1859. Translation from the *Bibliothek für Lager*, by Dr. W. D. Moore.

† *Opus citat.*, p. 376.

strong; it is remarkably liable to exacerbations from emotional excitement. Trousseau has, in one instance, seen projection of the precordium from long continued palpitation. The sounds of the heart are usually sharp and clear, as in ordinary nervous palpitation. A soft systolic murmur, more or less persistent, but rarely of long continuance when not dependent upon valvular lesion, is frequently heard at the apex of the heart. A murmur of similar quality and rhythm still more frequently exists at the base, either in the aorta or in the pulmonary artery; but it is not transmitted into the arteries of the neck. The former of these murmurs is most probably adynamic, the latter hæmic; both are occasionally harsh in quality. I have frequently heard, in these cases, *sounds* both single and double in the carotid arteries, but never murmur, where it was not due to pressure from the enlarged thyroid gland. Trousseau has observed, as a constant symptom in exophthalmic goitre, the "cerebral macula," viz., a reddening of the skin after slight irritation, as in cerebral or ataxic typhoid fever. Foot, likewise, noted this phenomenon in one of his cases.* An elevation of temperature of 1° to 2° F., during the paroxysm, has been noticed by Basedow and Teissier, and also by Cheadle.† These symptoms have been attributed by Trousseau to debility of the vaso-motor nerves. Menstruation is usually irregular and defective, but the three prominent symptoms of the disease are, in most cases, exhibited before the occurrence of this derangement. The order in which, relatively, the symptoms were exhibited in my 6 cases‡ is shown in the following table :

TABLE XV.—EXOPTHALMIC GOÏTRE.

Symptoms.	Order of Occurrence.				Total Cases.
	First.	Second.	Thirđ.	Fourth.	
Palpitation ...	4	0	1	...	5
Thyroid Enlargement ...	2	1	3	0	6
Projection of Eyes ...	0	4	1	1	6
Deranged Menstruation	0	1	1	4	6

So far as a conclusion may be drawn from these 6 cases, it

* *Irish Hospital Gazette*, vol. ii., p. 163.

† *St. George's Hospital Reports*, vol. iv., p. 183, *Ibid.*, vol. vii., 1875, p. 81.

‡ I have, unfortunately, mislaid the notes of the 7th Case, referred to at p. 490.

would appear that palpitation is most frequently the primary symptom, and next, in order of frequency, enlargement of the thyroid gland; that projection of the eyeballs is the secondary symptom in the great majority of cases; that thyroid enlargement is, but in a somewhat less proportion, the usual tertiary symptom; whilst derangement of menstruation constitutes, in most cases, the fourth symptom in the order of occurrence. The number is, however, obviously too small to furnish a reliable basis for induction.

The enlargement of the thyroid gland constitutes the most urgent symptom of the disease. It is usually slow but fitful in progress, the right lobe being, in most cases, the first portion affected, and attaining larger dimensions than the remainder of the organ.* Its primary increase is due to enlargement of the thyroid vessels; hence pulsation and *bruit de soufflet* during the period of vascular excitement, after which partial subsidence of the swelling is seen to occur. At a later period the parenchyma and the connective tissue of the gland are found to have undergone true hypertrophy; the organ is now more solid, and the increase of bulk has become permanent.

In some cases the excess of vascularity and the activity of pulsation of the thyroid body have been such as to lead to an error of diagnosis. Dr. Stokes has known the tumor to be mistaken for aneurism, a day having been actually appointed for the operation of tying the carotid artery before the error was detected.

Huskinness of voice, and even aphonia and stridor, are not unfrequently produced by the pressure of the enlarged thyroid upon the trachea and recurrent nerves.

Life has been actually endangered by mechanical pressure upon the trachea and blood vessels. Trousseau mentions the case of a boy in whom tracheotomy was contemplated as the only means of preventing suffocation; but relief was obtained by bleeding from the arm, the application of ice to the tumor, and the administration of digitalis.

I have, in one instance, seen the thyroid enlargement attain such

* The right lobe was larger than the left in 3 of Moore's 6 cases, and likewise in the case recorded by Banks. *Dublin Hospital Gazette*, vol. ii., p. 129.

dimensions, as to place the patient's life in the utmost danger from asphyxia and coma by pressure on the trachea and the jugular veins. The patient, a female, aged eighteen, was admitted into hospital under the care of my late colleague, Dr. Curran, on the 10th of January, 1870, with all the symptoms of Graves' disease well pronounced. The thyroid gland had already attained the size of half an ordinary melon; it pulsated violently, and, by pressure upon the jugular veins and the trachea, caused great congestion of the neck and face, and urgent dyspnœa. The eyes projected to an extreme degree, and the patient was unable to swallow solids of any kind. Ordinary treatment, including bleeding from the arm, was attended with only temporary relief. The swelling increased, the patient became delirious, and asphyxia was imminent. The tumor being remarkably tense, it was suggested in consultation that relief might be obtained by division of the cervical fascia. This operation, which was attended with no ordinary difficulty owing to the great number and size of the superficial veins, was successfully performed by my colleague, Mr. Hayes, on the 27th of February; the fascia was slit up upon a director along the mesial line, from the *fourchette* of the sternum to the os hyoides; the gland protruded through the wound, divaricating the edges of the incision to an extent of several inches, and the patient experienced immediate relief. The loss of blood did not exceed eight ounces.

On the following day the cervical fascia was further divided, and the sterno-hyoid and sterno-thyroid muscles, which were on the stretch, were partially cut through. Relief was now complete; the patient was enabled to lie down and to take solid food.

She was discharged on the 12th of April, six weeks and three days after the date of operation. The catamenia, which had been irregular, were re-established. The thyroid enlargement became soft, gradually diminished in size, and the wound cicatrized. The patient visited the hospital in May, 1871, viz., a year subsequent to operation. She was then in fairly good health, the thyroid tumor being not larger than a small orange.*

* Vide *Medical Times and Gazette*, May 27th, 1871. I saw this woman again in April, 1875. The thyroid enlargement then scarcely amounted to disfigurement, proptosis no longer existed, menstruation was regular, she felt quite well and was actively employed, but the pulse was slightly accelerated.

The appearance of the eyes constitutes the most striking characteristic of the disease; they are prominent and staring, as if under the continuous strain of a great physical effort, and impart to the face a maniacal expression. The pupils are, with few exceptions, usually dilated, and the conjunctival and sclerotic veins are more or less turgid. This condition of the eyes is exaggerated during a paroxysm of palpitation. It is usually symmetrical, but in a case recorded by Desmarres, one eye only was affected. Vision is in many cases misty, and the focal distance is usually shortened; but frequently there is no alteration of focus or of visual perception. Trousseau mentions a case, recorded by Pain, in which one of the eyes was actually dislocated from the orbit by pressure from behind.

The prominence and tension of the eyeballs have been attributed to three different causes; namely, excess of the vitreous humour; hypertrophy of post-ocular connective tissue, with cedema, or accumulation of fat; and dilatation and distention of the post-ocular vessels. In one instance Trousseau found the globes, which were of ordinary size, thrust out of the orbits by excess of areolar and adipose tissue.* In Basedow's and Keusinger's cases the eyeballs were atrophied, and the orbital fat was in excess.

As to the etiology of the disease, I consider Dr. Lauder Brunton's hypothesis the most tenable.† It is not only in consonance with the admitted condition of the carotid and thyroid arteries, but it explains the increase of ocular protrusion exhibited under vascular excitement, and the partial subsidence of the eyeballs which follows the cessation of a paroxysm. In a case recorded by Mr. Sharpley,‡ the retinal veins were seen to pulsate during a paroxysm.

After death, sanguineous extravasation has been found in the retina, the vessels of the choroid being congested. Pigmentary deposit in the retina has been detected in at least three instances; viz, in the case recorded by Withusen, in one examined by Dr. Argyll Robertson,§ and in a third case recently

* *Opus citat.*, p. 582.

† *Vide* p. 1051.

‡ *Medical Times and Gazette*, September 5th, 1874.

§ Dr. J. Begbie, *Contributions to Practical Medicine*, p. 175 (foot note).

under the observation of Dr. Nixon in the Mater Misericordiae Hospital.*

Anæmia, and in the female, leucorrhœa, are pretty constant symptoms of exophthalmic goitre. They are, however, by no means necessary, or even ordinary precursors of the "symptomatic triad," as suggested by Luton. In four of the cases published by Dr. Moore, anæmia did not exist; three of these patients were actually florid. Niemeyer does not admit a necessary connexion between anæmia and exophthalmic goitre.† Respiration is easily quickened, and not unfrequently it is permanently accelerated. There is insomnia, and the bowels are usually constipated. In the advanced stages the appetite for solids is indifferent, or entirely lost, and in many cases persistent vomiting exists. Intercurrent diarrhœa, epistaxis, intestinal hæmorrhage, and œdema of the feet, are occasionally witnessed in the course of the disease.

Doctor Graves has remarked the frequent association of hysteria with this disease; he has even suggested the dependence of the hysterical globus upon temporary enlargement of the thyroid gland. The connexion is, however, doubtful.

The disease is rarely fatal. When death results from it, dilatation of the heart with its consequences, congestion of the lungs, anasarca, and effusion into the serous cavities, is usually the immediate cause; but not unfrequently the patients succumb to intercurrent affections of the respiratory organs, to chronic renal disease, or to cerebral hæmorrhage. Death has likewise resulted from erysipelas, and from gangrene following anasarca of the lower extremities.

The recorded *post mortem* examinations are few. Besides those given by Trousseau, Cruise and McDonnell, Basedow, and Keusinger, already referred to, examples have been published by Marsh,‡ Smith,§ Withusen,|| Præhl,¶ Banks,** Reith,††

* This case has not been as yet published.

† *Opus citat.*, vol. i., p. 375.

‡ *Proceedings of the Pathological Society of Dublin*, 1841.

§ Stokes, *Diseases of the Heart and Aorta*, p. 291.

|| Trousseau, *opus citat.*, p. 570. ¶ *Ibid.*, p. 570.

** *Dublin Hospital Gazette*, June 1st, 1855.

†† *Medical Times and Gazette*, November 11th, 1865.

J. Begbie,* and Flint.† Other fatal cases are referred to by Cheadle‡ as having been recorded in Germany by Virchow and von Recklinghausen, and in France by Péter.

The case published by Dr. Banks claims a more extended notice, as being one of the most complete on record. The patient, a female, aged thirty, when fifteen years old, suffered from mental disquietude and emotional excitement. Her menstrual function had been always irregular, and for the last twenty months of her life it was entirely suspended, having been, about that time, suddenly arrested in the midst of a period. She had been subject to palpitation, and in the winter to bronchitis.

The eyes were prominent, brilliant, and staring; but they were not congested, nor was vision in any way affected.

The thyroid gland was much enlarged, the right lobe in a greater degree than the left; it increased in size during palpitation and fits of coughing, and it yielded a purring thrill, and a loud continuous venous hum. There was arterial throbbing in the neck, accompanied by a single and interrupted arterial sound.

The pulse was rapid, feeble, and unequal; it was likewise irregular and intermittent. Precordial dulness was extended, and the action of the heart was irregular and tremulous, but no cardiac murmur existed.

Towards the close of the patient's illness there was œdema of the lower extremities and eyelids; the urine contained albumen and some blood, and, in the recumbent posture, there was great respiratory distress whilst the cervical throbbing and murmur became nearly imperceptible. Death occurred by syncope. The *post mortem* appearances I shall record in the author's words:

"The thyroid gland, enlarged to four or five times its natural size, was found to cover, to a considerable extent, the front of the trachea. The right lobe was larger than the left; the thyroid veins were remarkably dilated; the gland was dense, very

* *Dublin Hospital Gazette*, vol. ii., p. 109, from Case Book of *The Edinburgh Medical and Surgical Journal*.

† *Diseases of the Heart*, third edition, p. 311.

‡ *St. George's Hospital Reports*, vol. iv.

solid to the feel, and lobulated. A section of different parts disclosed the existence of numerous cysts, containing a yellow fluid like honey. The contents of some of the cysts were dark coloured, and resembled coagulated blood. The microscopic appearances were similar to those observed and figured by Rokitsansky in ordinary enlargement of the thyroid.

"The jugular veins were enlarged. The bronchial glands were found of an unusually large size. The heart was enlarged generally, and the cavities dilated, but not to any considerable extent.

"The heart was as large as that of an ordinary man, the subject being a woman under the middle size. The valves were free from disease, with the exception of slight thickening of the anterior edge of the mitral valve.

"The lungs were highly congested, and the bronchial membrane bore the marks of intense inflammation. The liver appeared as if in the earliest stage of cirrhosis; the spleen large and congested.

"The brain softer than natural; the lining membrane of the ventricles much thicker than usual. The kidneys had undergone the changes usually observed in the early stages of Bright's disease."

In Dr. Begbie's case, which was that of a man, aged thirty-one, in which all the symptoms of the disease existed, death occurred from congestion of the lungs and general serous effusion. The heart was dilated and thinned; it was distended with fluid blood. The jugular veins were dilated, that of the left side being an inch and a-half in diameter; the thyroid was enlarged; the spleen and kidneys were likewise enlarged; the liver was cirrhotic.

I am indebted to Dr. Cruise for the following report of the case, already referred to, which came under his notice: "Margaret M., aged thirty-six years, mother of several children, entered the Mater Misericordiæ Hospital under my care, September 26th, 1864. Her case was a well marked one of exophthalmic goitre. The disease was of a few months' duration, and came on while wet nursing a child in Lord L.'s family. From the time of her admission, up to her death, November 30th, 1864, she steadily sank. Palpitation was most violent, and uncontrolled by the

remedies used (*viz.*, digitalis, bromide of potassium, iodine, and cold to the thyroid, as advised by the late Dr. Aran of Paris). During the last few days of her life she passed into a semi-conscious state, with sordes on the teeth and gums, in fact a typhoid state, with remarkable dilatation of the pupils, which contracted markedly with the contraction which existed at first. On examination after death, the heart was found slightly hypertrophied, but otherwise sound. The thyroid was enlarged, but not to the extent observed during life, and the eyeballs were healthy. The only marked change discovered was in the sympathetic nerve in the neck, the ganglia of which, on both sides, were so atrophied as to be barely distinguishable from the shaft of the nerve. On examination under the microscope (made by Dr. Robert McDonnell) the ganglia were found to consist of little more than connective tissue and fat, none of the ganglionic structure being visible."

In Dr. Reith's case, the middle and inferior cervical ganglia of the sympathetic were enlarged, hardened, and infiltrated with granular matter; the nerve itself between these ganglia was likewise enlarged, and the surrounding cellular tissue was thickened. Dr. Reith was disposed to regard these changes as essentially strumous.

A question of great importance, as bearing upon the pathology of exophthalmic goitre, is the precise state of the heart in its advanced stages.* Towards the settlement of this question morbid anatomy has contributed but very scant information. In all the recorded autopsies, one or both ventricles were found to be dilated or hypertrophied; but, in every instance, with the exception of those by Drs. Banks and Cruise just given, valvular or aortic disease, adequate to account for these conditions, likewise existed. Hence, clinical observation constitutes almost the only available evidence from which a conclusion can be drawn.

The apex of the heart has been displaced outwards in every chronic example of the disease. In most of the recorded cases the first sound of the heart was sharp and clear, whilst the radial pulse was feeble. In one of my cases (No. 132, p. 1068) reduplication of the second sound existed without pulmonary con-

* This subject has been already discussed at p. 489.

gestion, whence I inferred dilatation of the left ventricle. From these data, the conclusion may, I think, be legitimately drawn, that the left ventricle had become in some degree dilated. I am, therefore, in agreement with Dr. Stokes on this question; and I think the evidence to the contrary adduced by Trousseau is far from conclusive. Such is, likewise, the opinion of Virchow and Niemeyer. I cannot concur in the opinion of M. Luton, that the right side of the heart is always passively dilated in this disease;* nor even if such were the case, could I admit his conclusion that the symptoms which characterize the affection would be explained thereby.

The *diagnosis* of exophthalmic goître, in its typical form at least, cannot be attended with difficulty. It is liable to be confounded with ordinary bronchocele only; but from this it differs, as pointed out by Stokes, in not being connected with soil or climate, and in the remarkable variations of volume exhibited by the tumor. The enlargement of the thyroid, moreover, seldom attains to the magnitude exhibited in bronchocele; and, as remarked by Graves, it becomes stationary at a stage when the growth of bronchocele is accelerated. The swelling does, however, contrary to the opinion of Graves, not unfrequently "amount to actual deformity." The remarkable throbbing of the carotid arteries usually witnessed in this disease, may be confounded by a careless observer with that due to inadequacy of the aortic valves; but the absence of a diastolic murmur at the base of the heart, and the character of the radial pulse, will suffice to exclude the latter affection. The prognosis is, on the whole, not unfavourable, but it must be made with reference to the actual condition of the patient. Most of these cases improve and relapse alternately, the disease becoming chronic, and ultimately stationary. Death seldom results directly from it.

The *treatment* must vary, according to the view taken of the pathology of the disease, and the special indication afforded by symptoms.

The resemblance which, in one feature, it bears to bronchocele, has suggested the use of iodine, and several examples of cure by

* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques*, tom. xvi*, p. 506.

means of this agent have been recorded.* Trousseau, however, believes that iodine is positively injurious. Iron may be given with benefit where anæmia predominates, if the pulse-rate be not excessive.† In the latter case, especially if the pulse be at the same time weak and intermittent, the impulse of the heart feeble, and the first sound clear but faint, digitalis is the appropriate agent to be employed. Under its use, in doses of ℞ to ℞xv of the tincture given thrice daily, with the spirit, or compound tincture, of chloroform, the pulse-rate is rapidly reduced, whilst the action of the heart acquires strength and steadiness. Digitaline may be substituted, in doses of one-fortieth of a grain every sixth hour till the above mentioned effects are exhibited, or, should the stomach prove intolerant of the drug in any form, 3j of the *unguentum digitalis* of the *Hamburgh Pharmacopœia* may be smeared over the precordium night and morning, the result being carefully observed. In every case the use of the drug should be suspended when the pulse has been reduced below 100 in the minute. Trousseau, however, thinks digitalis should be given till the pulse-rate falls to 70, and the earliest symptoms of its toxic effects are exhibited; viz., vertigo and nausea. This is unnecessary, and not free from danger. It is, moreover, objectionable, as precluding the repeated use of the medicine, without which permanent benefit cannot be obtained.

Aconite may be given with advantage during the paroxysms of palpitation, if the impulse of the heart be vigorous and the pulse free from intermission. Bromide of potassium may be likewise administered with benefit in such cases.‡ Gelseminum, arsenic, and iodo-bromide of calcium§ have been used in the treatment of exophthalmic goitre; galvanization of the cervical sympathetic, as first resorted to by Dusch, has been likewise tried, and with satisfactory results, by Chvostek, Moritz Meyer, and by Eulenburg and Guttmann.||

One of the primary objects of general treatment in cases at-

* *Vide Stokes, opus citat.*, and Chendle, *St. George's Hospital Reports*, vols. iv. and vii., also Case 134, p. 1071.

† *Vide Foot, Irish Hospital Gazette*, vol. ii., p. 165, and Sharpley, *locus citat.*

‡ *Vide Case 130*, p. 1066.

§ Dr. Guptil, *American Journal of the Medical Sciences*, January, 1874.

|| *Die Pathologie des Sympatheticus*, 1873.

tended with derangement of menstruation, should be to re-establish this function. Improvement generally coincides with the return of the menstrual flux at the monthly periods, and, as remarked by Charcot,* such is likewise the case on the occurrence of pregnancy.

Cold water bathing, and especially cold showers, at the proper season, and during the intervals between the monthly changes, are of great value as a means of allaying nervous excitement and promoting general nutrition.

Bleeding from the arm has been followed by complete relief during a paroxysm of dyspnoea in which strangulation from vascular engorgement of the thyroid body had been imminent. Ice should be applied over the tumor, at the same time, as suggested by Aran and Trousseau.†

In extreme cases, tracheotomy has been suggested as the only means of saving life; but, irrespectively of the unfavourable prospect of ultimate recovery which this operation affords, even though successful in rescuing the patient from immediate death, it is attended with the utmost difficulty and danger in the performance, owing to the enlargement and great vascularity of the thyroid body. I have, in a previous page (1057) indicated an alternative procedure, which was eminently successful in the only instance in which it has been resorted to; namely, division of the cervical fascia over the tumid gland.

The following six cases constitute the only examples of exophthalmic goître of which I have preserved full notes.

CASE CXXIX.—*Exophthalmic Goître and Impairment of Vision; Palpitation, with Murmur in the Pulmonary Artery; Rapid Pulse; Irregular Menstruation.*

Anne C., aged thirty years, visited the Mater Misericordiæ Hospital as an extern patient, May 31st, 1867. Three years previously she had been in hospital for pain in the left shoulder. About the same time she noticed a swelling in her neck, and her sight began to fail. During the last year menstruation had been

* *Gazette Hebdomadaire*, 1862.

† *Bull. de l'Acad. de Médecine*, December 4th, 1860, tom. xxvi., p. 122.

irregular, and on two occasions it was suspended for two months. The eyes were prominent, and vision was imperfect in the left; it was "misty" and "cloudy" after exertion. There was general enlargement, with pulsation of the thyroid gland. The carotid arteries pulsated visibly; the pulse was 168 and weak, and the sounds of the heart were sharp, clear, and free from murmur. She was directed to take, twice daily, gr. ss of extract of belladonna, with an equal quantity of extract of nux vomica.

June 15th, 1868. She again presented herself in the out-patients' room, and the following note was taken. Pulse small, 160; heart's action "hammering," and in rate corresponding to the radial pulse, but not attended with murmur. Thyroid enlarged and pulsating; eyes prominent, and sight weak; respiration 36; has cough, and is remarkably nervous.

October 7th, 1868. The report made was as follows. Pulse 168; *bruit de râpe* over pulmonary artery, but not audible elsewhere. Menstruation has been suspended for the last two months; is deaf in right ear. To have bromide of potassium, grs. x, thrice daily.

January 13th, 1869. The last note made of this case bears the above date, and is to the following effect. Pulse 150; eyes prominent; thyroid large and pulsating; heart bounding.

It is noteworthy that impairment of vision not only existed in one eye, but was coeval with protrusion of the eyeballs, and was one of the earliest symptoms observed.

CASE CXXX.—*Exophthalmic Goitre; Palpitation, with Roughness of the First Sound, and Throbbing of the Carotids; Irregular Menstruation; Improvement.*

Eliza E., aged twenty-two years, a domestic servant, of fair complexion, and a native of Birmingham, visited the extern department of the Mater Misericordiæ Hospital on the 10th, and again on the 17th of January, 1868. She had been for several years suffering from palpitation, which first occurred after severe mental affliction. Six months previous to her visit she noticed that her eyes projected, and three months later, that her neck

was swollen. Pulse 132; eyes remarkably prominent and staring especially the left, which was myopic. The thyroid body was enlarged, and pulsated strongly. There was palpitation of the heart, with roughness of the first sound, and visible pulsation of the carotids accompanied by a systolic arterial murmur. Menstruation was irregular, the periods varying from four to six weeks. Appetite, sleep, and state of bowels natural; tongue clean. To have grs. viij of bromide of potassium thrice daily. A few months later this girl reported herself as greatly improved, and able to resume her employment.

Palpitation preceded the other two events in this case, and protrusion of the eyes was second in order.

CASE CXXXI.—Palpitation; Exophthalmia; Enlargement and Great Congestion of Thyroid Body; Systolic Murmur in Thyroid Gland and in Carotid Arteries; Dyspnoea; Dysphagia; Cough and Hæmoptysis; Œdema of Feet, and Occasional Syncope; Rapid Pulse; Intermittence of the Heart; Masking of the Second Sound; Extension of Precordial Dulness, and Displacement of Apex Outwards.

Margaret M., aged forty years, a dressmaker, married, and has had nine children, is still "regular," visited hospital, July 6th, 1869. Her illness commenced with palpitation eight years previously, and about the same time her eyes became somewhat prominent. Four years later her breathing became short, and about the same time she began to experience some difficulty in swallowing solids. For the last two years she has been suffering from cough with mucus expectoration, which, on three several occasions, was mixed with blood. Epistaxis has occurred occasionally. During the heat of the present summer she had been prevented, by dyspnoea, from assuming the recumbent posture, and for the last fortnight she has been drowsy.

When first seen by me her condition was the following: The eyes were remarkably and equally prominent, conjunctivæ congested, and pupils contracted. She possessed the power of closing the eyes by an effort, but during sleep they remained open. Both lobes of the thyroid body were greatly enlarged, and there

was congestion of the veins of the neck, which was much increased when the patient coughed. Indeed, she declares that during the act of coughing, the tension is so great over the thyroid gland, that she feels as if it must "burst." The root of the neck was tumid and rounded off. She did not suffer from headache. Bowels confined. Respiration was shallow, and 60 in the minute. The pulse was 108, and weak. The action of the heart was characterized by occasional intermissions; the sounds were free from murmur, but the second was not clear at any point of the precordium. The respiratory sounds were weak, but equal on both sides. Percussion-resonance normal. To have aperients, and grs. x of bromide of potassium, thrice daily.

July 30th. She has had slight weakness, with temporary loss of vision and of consciousness, two or three times daily for the last fortnight; this was not caused by exertion.

April 6th, 1870. She has been "irregular" for the last six months, and the feet have been swollen at night for the last four months. Pulse 120. Eyes prominent and lachrymose, and when she coughs forcibly she feels them tense, and as if she could not close them.

The thyroid body was much enlarged, and crossed by turgid veins; it yielded a loud systolic bellows-murmur. A murmur of similar character and rhythm was audible in the carotid arteries. The face was congested. Precordial dulness was extended; apex-pulsation in nipple-line, and impulse strong.

This patient ceased to attend at the hospital, and I lost sight of her.

CASE CXXXII.—*Palpitation and Rapid Pulse; Exophthalmia; Enlargement and Pulsation of Thyroid; Throbbing of Carotids; Derangement of Menstruation; Abrupt Cardiac Impulse; Sounds Clear, and Free from Murmur; Second Sound Reduplicated at Midsternum; Dilatation of Left Ventricle.*

Miss McC., a farmer's daughter, aged twenty-three years, consulted me on the 13th of May, 1870. Had undergone much labour in milking cows at a large dairy. Two years previously she began to experience palpitation; about the same time she

noticed swelling at the root of her neck, and enlargement of her eyes, both of which changes have been gradually increasing since that date. Has had, for the preceding six months, slight tenderness of the left knee, at a point some distance above the joint.

When she came under my notice, her skin was dusky and her eyeballs were prominent, giving her a remarkable staring expression, which her brother, who accompanied her, characterized as a "queer look." The thyroid body was enlarged and pulsatile, but, as I was informed, it varied inversely as the activity of the menstrual function. There had been no "change" for the preceding two months, and the thyroid was, at the time of examination, remarkably large. Pulse 144, and regular; throbbing of the carotid and radial arteries; the impulse of the heart was abrupt and "hammering"; the sounds were clear and unattended with murmur, the second being reduplicated at midsternum.

To have bromide of potassium (grs. x) thrice daily, and compound aloetic pill (grs. v) every night. To give up the use of strong tea, to which she had been addicted. I have had no further report of this patient. In the absence of the signs of pulmonary congestion in this case, I regarded reduplication of the second sound as evidence of dilatation of the left ventricle. The clear and sharp character of the sounds tended to confirm this view.

CASE CXXXIII.—Palpitation; Exophthalmia, and Enlargement of Thyroid Body, with Pulsation, Thrill, and Systolic Bruit; Bruit de Diable; Weak and Rapid Pulse; Tumultuous Action of Heart, with Systolic Murmur both at Apex and Base; Amenorrhœa; Great Dilatation of the Pupils; Emaciation; Temporary Improvement, and Relapse.

Doctor Stokes was kind enough to invite me to see, with him, a remarkable example of this disease, on the 28th of April, 1871. The patient was a young lady from the country, aged twenty-four years. She gave the following history of her illness: Two years previously she had been much frightened, and immediately afterwards she began to suffer from palpitation. About a week subsequently her eyes began to project, and the root of her neck

to swell. Her menstrual function now became deranged; she became nervous and sleepless, and began to lose flesh.

Under a course of treatment with tonics and chalybeates she improved considerably; the swelling of the thyroid subsided, the eyeballs receded, the palpitation was mitigated, and menstruation was re-established.

This improvement was of brief duration, and when I saw the patient her condition was as follows: She was greatly emaciated, pale, and nervous; the eyeballs projected quite beyond the lids; she closed the eyes with great difficulty, the effort to do so being attended with circumorbital pain, and she slept with the eyes open. The pupils were greatly dilated, and when the patient sat with one side of her face to the window, light entering by the pupil and reflected from the *fundus oculi*, was distinctly transmitted at an angle through the sclerotic.

The thyroid body was much enlarged, especially on the right side, and coursing over it were seen large and distended veins which pulsated synchronously with ventricular systole; this pulsation was arrested by light finger-pressure at the root of the neck.

A loud intermittent *bruit*, of a buzzing character, and synchronous with ventricular systole, was audible over the thyroid body, and at its posterior margin a combination of triple sounds, consisting of carotid *bruit de choc*, jugular *bruit de diable*, and the thyroid buzz just described, was heard. The thyroid body pulsed sensibly, and communicated to the hand a faint thrill. Pulse rapid and weak; cardiac impulse strong and heaving, and both at apex and base a faint but rough systolic murmur existed. The apex-pulsation was in the nipple-line. She had not menstruated for the last four months. The bowels were regular.

Small doses of citrate of iron and quinia were prescribed, and inunction of the precordium with the ointment of digitalis (Hamburgh Pharmacopœia) was directed. I had not again an opportunity of seeing this lady, and I have heard nothing further of her case.

CASE CXXXIV.—*Menorrhagia ; Palpitation and Rapid Pulse ; Great Enlargement of Thyroid Body, and Extension of Swelling into Anterior Mediastinum ; Enlargement and Distention of Cervical, Thoracic, and Brachial Veins ; Throbbing of Carotid Arteries ; Systolic Bruit de Soufflet in Thyroid ; no Exophthalmia, but Temporary Obscuration of Vision ; Improvement under Treatment with Iodine.*

Miss O'B., aged about forty years, consulted me in September, 1872, She had menstruated regularly, but with pain. Her neck began to swell six years previously, but from this she felt no inconvenience till nine months prior to her visit to me. When first noticed, the swelling of the neck was chiefly on the right side.

In January, 1872, she was suddenly attacked with "flooding," which lasted continuously for twenty-four days, the discharge, consisting partly of liquid, and in part, of clotted blood. The loss was very considerable, and greatly reduced her strength. After this occurrence she began to suffer from palpitation and dyspnoea on slight exertion, and the swelling in the neck rapidly increased.

At the date of this lady's visit to me the thyroid body had attained a large size, and, as judged by the percussion-dulness and the prominence of the sternum, a tumor continuous with it extended fully two inches into the anterior mediastinum.*

The superficial veins at the root of the neck were greatly enlarged and tortuous, the external jugular being quite as large as the internal jugular in its normal state, and from the thyroid cartilage to the *fourchette* of the sternum a vein of equal size coursed vertically along the middle line. Over the thyroid body generally a loud systolic *bruit de soufflet* was audible. The cervical arteries pulsated visibly, and the superficial veins of the arms and of the upper portion of the chest on both sides were enlarged and distended. The upper portion of the sternal region, for about two inches downwards, was prominent and dull on percussion as already stated, whilst both subclavicular regions were

* A case, which ultimately proved fatal, exhibiting a similar extension of the thyroid into the chest, is reported from Guy's Hospital, in the *Medical Press and Circular* of March 23rd, 1870. The heart was normal in this case.

depressed, and, in the latter situations respiration was feeble. Over the sternal prominence a loud musical stridor was heard with both inspiration and expiration.

The heart pulsated in the normal position; its action was rapid and tumultuous, but the impulse was rather abrupt than strong, whilst the second sound was sharp, clear, and free from murmur.

The pulse was regular but abrupt, and in the sitting posture it averaged 144. The eyes were not prominent, but on the occasion of her third visit, viz., on the 1st of October, she experienced a mist passing over her eyes from below, when she looked upwards. There was then slight cedema of the feet and of the right arm and shoulder.

The slightest exertion caused much difficulty of breathing. Sleep was disturbed, and she experienced some discomfort at the root of the neck when swallowing solids. Voice husky, and singing voice entirely lost. Face of a leaden hue. To have grs. v of iodide of potassium, with ℥ij of tinct. of iodine, thrice daily, and the tumor to be painted with the liniment of iodine. To have warm sea-baths, and subsequently open sea-bathing. Digitalis had been given in large doses, with very little benefit.

I saw this lady again in the following year. She had continued the iodine treatment during the interim. The thyroid swelling was now much reduced, and the venous engorgement less. She had not had a return of the flooding; her monthly health was now regular, and her strength had improved. I have not since heard of her.

CHAPTER X.

DISEASES OF THE AORTA.

THESE diseases may be classified under the three heads of

- (a) Atheromatous change, in its several stages.
- (b) Simple dilatation.
- (c) Aneurism.

Atheromatous transformation of the coats of arteries is a result of malnutrition, and essentially degenerative. Not unfrequently the products of a low form of inflammation of the internal and middle coats are combined with those of tissue degeneration. Moxon says, "Atheroma is in continuity with arteritis, and graduates from a condition in which no inflammatory results can be found, into one in which inflammation is unmistakably present." He holds that inflammatory irritation of arteries arises from vascular tension, and remarks that diseases which lessen the volume of the blood, and therefore diminish the tension of the arteries, such as phthisis and mitral obstruction, are very rarely associated with atheroma.* Holmes considers strain of the walls of arteries to be the principal factor in the production of atheroma.†

Niemeyer maintains that antecedent chronic inflammation is a necessary condition of atheromatous change.‡ Virchow is of the same opinion; adding, that the primary irritation results in enlargement of the connective-tissue corpuscles, multiplication of nuclei, cell-proliferation by endogenous growth, and, finally, fatty degeneration of these new products.§

The degenerative changes in the coats of blood-vessels constituting atheroma, consist essentially in fatty and calcareous meta-

* *Guy's Hospital Reports*, vol. xvi., third series, 1871, p. 431.

† *System of Surgery*.

‡ *Text-Book of Practical Medicine*, vol. i., p. 345.

§ *Cellular Pathology*, translated by Chance, p. 361.

morphoses, as shown by Gulliver,* and admitted by Paget,† Erichsen,‡ and all modern pathologists. Aitken regards atheroma as a fatty product of degeneration of tissue; but he considers rheumatism, gout, and syphilis, to be the ordinary causes of this change.§ Hodgson has noticed the frequent occurrence of aneurism in persons who had suffered from syphilis and taken large quantities of mercury.|| The doctrine of syphilitic causation of atheroma is, however, very questionable. It is noteworthy, that whilst syphilis is more common in civic than in rural populations, aneurism is not proportionately in excess amongst the former; and, as remarked by Mr. Cooper Todd, whilst syphilitic affections were formerly more common and more virulent amongst soldiers than at present, aneurism has been latterly rather on the increase in the army.¶

The influence of gout in the production of atheroma cannot, I think, be doubted. The degenerative changes, which, in chronic gout, are exhibited by the several structures in and around the joints, are kindred to, and most frequently associated with, corresponding changes in the walls of the arteries. I cannot recognise rheumatism as a cause of atheroma, except in so far as it may be associated with gout, under the form of "chronic rheumatic arthritis."***

In many cases atheroma is directly and exclusively the result of persistent arterial tension from hypertrophy of the left ventricle.†† Hence, its frequent association with the cirrhotic form of Bright's disease, and with patency of the aortic valves. Hence, likewise, it is first exhibited in the arch of the aorta, which is most exposed to pressure from an hypertrophied ventricle. The change commences in the middle or fibrous coat, in the form of minute fawn-coloured spots; these gradually extend and coa-

* *Medico-Chirurgical Transactions*, vol. xxvi., p. 86.

† *Lectures on Surgical Pathology*, vol. i., p. 138.

‡ *Science and Art of Surgery*, 1869, vol. ii., p. 1.

§ *The Science and Practice of Medicine*, fifth edition.

|| *A Treatise on the Diseases of Arteries and Veins*, 1815.

¶ *Army Medical Reports*, 1868.

*** *Vide A Treatise on Chronic Rheumatic Arthritis*, by Robert Adams, M.D., 1857.

†† *Vide* p. 513, *et sequent.*, and p. 824.

lesce, assume a deeper yellow tint, and become more dense and brittle. In the same proportion, the coats of the arteries lose their properties of resistance and elasticity, and the vessel is slowly and permanently dilated. The middle coat thus deteriorated may be rent by the pressure of the blood, or it may undergo the further change of calcification, forming scales, the sharp edges of which, as observed by Richet,* may wound the "intima," and thus give rise to aneurism. The internal coat, when unsupported on the outside, from yielding or disintegration of the "media," is, in the great majority of cases, quickly broken through, and aneurism is the necessary result. The process of change above sketched, may, however, not extend beyond the stage of dilatation of the vessel. In the case of the superficial and smaller arteries, such as the radial and temporal, owing to their loose connexions and the tenuity of their walls, this is always accompanied by elongation and inflection.

Dilatation of the aorta, carried to a certain point, and involving the orifice of the vessel, if unattended with proportionate expansion of the valves, will give rise to patency and a murmur of reflux,† which, if the degree of inadequacy be extreme, will be prediastolic in rhythm.‡ If the internal surface of the vessel be rough from atheromatous change, a systolic murmur, located above the base of the heart, and not transmitted into the arteries of the neck, will be the result.§ I cannot admit that equable and moderate dilatation of the aorta is cognizable from physical evidence in the absence of the conditions and attendant signs just specified.

Dilatation of an artery carried to an extreme degree, and unaccompanied by breach of continuity in one or more of its coats, constitutes true aneurism; a form of the disease denied by Scarpa,|| but admitted by Corvisart,¶ Bertin,** Laennec,†† and

* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques*, 1865, vol. ii.

† *Vide* Case 87, p. 866.

‡ *Vide* p. 843, and Case 98.

§ *Vide* p. 839, and Case 62.

|| *A Treatise on Aneurism*, Wishart's translation, p. 54.

¶ *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels*, Hebb's translation, p. 302.

** *Traité des Maladies du Cœur*, p. 93, *Observations* 37 and 38.

†† *Traité d'Auscultation Médiate*, tom. ii., p. 688.

by most modern authors. True aneurism must be recognized, but only as representing a state of transition; the size attained and the figure presented by it depending upon the degree and extent of antecedent disease in the walls of the vessel, and the strain exercised upon them. The distinction between dilatation and true aneurism, as recognized by modern writers, has reference rather to the figure than the degree of enlargement; a fusiform expansion of the vessel over a limited portion of its length, or a yielding of a portion of its circumference in the form of a sacular protrusion of all its coats, constituting true aneurism, whilst an equable enlargement, formerly designated cylindroidal aneurism, is now, according to its degree, recognized as dilatation of the vessel.

Aneurism, therefore, as I understand it, is either *true*, by which I would imply a localized bulging or protrusion of all the coats of the artery; *false*, one or more of the tunics being broken through; *compound*, where a false is engrafted upon a true aneurism by outgrowth from a limited portion of its surface;* *dissecting*, constituted by rupture of the internal and middle coats, the blood having formed a new channel by separation of the outer from the middle coat over a limited portion of the circumference of the artery, and re-entered the vessel by a second opening, more or less distant from the original rent; and *varicose*, consisting in a communication between an aneurism and a vein, the pulmonary artery, or one of the chambers of the heart.†

False aneurism, the form in which the disease is most frequently presented, is usually the result of laceration or fissuring of the internal and middle coats of the artery, already deteriorated by atheromatous change. The blood insinuates itself through the rent so formed, and by its pressure determines the development of aneurism. Occasionally, as observed by Corvisart,‡ aneurism results from perforation of the inner coats of the

* Vide Corvisart, *opus citat.*, p. 302, where an example is given.

† I do not propose discussing aneurism in its surgical aspects, and therefore I have not included in the above category, traumatic aneurism, aneurismal varix, or aneurism by anastomosis.

‡ Hebb's translation, p. 276.

vessel, by the rupture of an abscess situate between the middle and external coats. Laennec also admits this mode of origin of aneurism.* False aneurism may be circumscribed or diffused. Circumscribed false aneurism has been divided into two species; namely, "mixed internal," and "mixed external." The "intima" protruding through a rent in the middle and external coats, and with the connective tissue forming the sac of the aneurism, constitutes the former species, the identity of which rests upon the authority of Dubois and Dupuytren based upon a single observation, and is now not admitted. The "mixed external," or the "aneurism of transition" of Cruveillier, is produced by rupture of the internal and middle coats of a previously diseased or dilated artery, the external coat, supported by the connective tissue, alone forming the wall of the sac. This is the most common form of aneurism, and that under which, except where other forms are expressly specified, the disease will be discussed in the succeeding pages.†

Diffuse false aneurism consists in an accumulation of blood outside the artery by rupture of all its coats, and limited only by the surrounding parts. It arises either from wound or laceration of the vessel by mechanical violence, or from rupture of the sac of a mixed external aneurism.

Aneurism is essentially, though not exclusively, a disease of middle age. Out of 120 cases reported by Lisfranc, 59 occurred between the ages of thirty and forty-five years;‡ and of 551 cases collected by Dr. Crisp, 398 were represented as having occurred between the ages of thirty and fifty years.§

Of 15 cases of aneurism of the aorta which have been under my observation and treatment, 11 were between the ages of thirty and fifty years, 3 were over fifty, and one only was under thirty years. Tables XVI. and XVII. comprise 84 cases of aneurism of the aorta; of these, 55 were between the ages of

* *Traité d'Auscultation Médiate*, tom. ii., p. 688.

† Dr. Crisp (*Diseases of the Blood Vessels*, p. 108) mentions a variety of false aneurism formed by projection of the internal and middle coats through a rent in the outer tunic, and refers to an example of this kind in the Museum of the London College of Surgeons (No. 1642).

‡ Reichet, *opus citat.*

§ *Opus citat.*, p. 113.

thirty and fifty years, 10 were over fifty, and 4 were under thirty years; in 15 instances the age is not stated. The principal cause of the prevalence of aneurism at middle age must be the vigour and activity of the body at this period of life. Atheromatous change and calcification of the arteries is most common after the age of sixty, as stated by Bichat.* But the smaller percentage, and the less degree of arterial change at an earlier period of life, are more than counterbalanced by the vascular strain arising from greater muscular power and habitual physical effort in middle age.

Sex has a manifest influence in the production of aneurism. Of the 551 cases referred to by Crisp, less than one-eighth had occurred amongst females, whilst my tables show only 10 females out of 84, or a little over one-eighth. The more laborious habits, and the greater muscular vigour of the male as compared with the female sex, by which vascular tension and strain are produced, may afford a satisfactory explanation of this difference. Mr. Porter has denied the influence of labour and muscular effort in the production of aneurism;† but, notwithstanding his great eminence as an authority on this subject, I cannot concur in the opinion just stated. Occupation, when ascertained in regard to a sufficient number of cases, may be accepted as a criterion by which to form a judgment on this question. My conclusions shall be drawn from examples of aneurism of the aorta exclusively; but manifestly they must apply equally to aneurism of the minor arteries.

Of 57 cases in Tables XVI. and XVII. in which the occupation of the patients is given, 12 were mechanics, 10 were soldiers or pensioners, 8 were labourers, 5 porters, 4 cabmen, 4 gentlemen, 4 housekeepers (females), 2 grooms, 2 policemen, 1 was a butler, 1 a sailor, 1 a dairyman, 1 a cattle drover, 1 a coal heaver, and 1 a strolling player upon a wind instrument. Thus, it would appear from the statistics at my disposal, that mechanics, soldiers, labourers, and porters, men whose avocations demand frequent and extreme muscular effort, or prolonged exercise under unfavourable conditions, enjoy an unenviable dis-

* *Anatomie Générale*, tom. ii.

† *Observations on the Surgical Pathology and Treatment of Aneurism*.

tion in regard to liability to aneurism of the aorta. Indeed, in most instances, the first symptoms of aneurism can be traced to a definite occurrence, involving great strain or effort, or to a severe shock.

The relative frequency with which different arteries are affected with aneurism, would appear to depend upon (a) their magnitude and proximity to the heart, and (b) their liability to strain or injury, whether by the force of the blood current, or from mechanical causes. Hence the disease is most common in the aorta. It has occurred with about equal frequency in the innominate, carotid, subclavian, iliac, femoral, and popliteal arteries.*

The greater liability of the aorta, as compared with other arteries, arises from its immediate exposure to the force of impulsion from the left ventricle. Owing to this circumstance, it is not only more liable to undergo those structural changes of its walls which predispose to aneurism, but such changes having been effected, it is more likely to suffer disintegration of structure from the force of the blood current. The figures representing the relative liability to aneurism of the different portions of the aorta, as deduced from the 84 cases comprised in Tables XVI. and XVII., are as follows : Ascending portion of arch, 30 cases ; abdominal aorta, 16 ; transverse portion of arch, 14 ; thoracic aorta, 8 ; descending portion of arch, 3. Thus, the frequency of aneurism of the ascending portion of the arch of the aorta was not only in excess of that in any other portion of the vessel, but equal to that of its occurrence in any *two* other portions of it. Two or more portions of the vessel were affected in the following relative frequency : Ascending and transverse portions of arch, 8 ; transverse and descending portions of arch, 3 ; ascending portion of arch and thoracic aorta, 2 ; transverse and descending portions of arch and thoracic aorta, 1 ; entire arch, 1 ; ascending portion of arch and abdominal aorta, 1.

As to the pathology of aneurism, I will only remark here, that I believe structural alteration of the coats, in a greater or less degree, always precedes simple or aneurismal dilatation of an artery ; and that vascular strain or tension is the immediate or

* *Vide* Hodgson, *op. cit.* ; Lawrence, *Lectures in Lancet and Medical Gazette* ; Chomel, *Dictionnaire de Médecine* ; and Wardrop, *Cyclopædia of Practical Surgery*.

determining cause of its enlargement.* Habitual vascular tension leads to impairment of nutrition, degenerative change, and loss of elasticity in the middle coat of the artery. Such changes being established, the rate of expansion of the disorganized portion of the vessel depends upon the degree of internal pressure to which it is subjected, and the frequency of repetition of such pressure. A high degree of blood-pressure suddenly brought to bear upon an artery in an early stage of structural alteration, may, within the period of a single act of respiration, produce the same result as a less degree of tension, exercised at intervals for many months, upon a vessel in a more advanced state of disease.

Where aneurism is the result of momentary strain, great muscular effort, or violent succussion of the chest or back, it is always of the false variety, and the patient is sensible of the occurrence of an accident, as of "something giving way" within the chest or abdomen. Sharp pain is felt at the seat of injury, the breathing is usually quickened, hæmoptysis not unfrequently occurs, and a sense of faintness, sometimes amounting to actual syncope, is experienced. In many instances in which the subsequent history of the case has proved the occurrence of rupture of some of the coats of the vessel at the moment of the imputed accident, the injured person has quickly recovered from the shock, and experienced but little inconvenience for months afterwards.† In all such cases, however, an appreciable deterioration of health, consisting in one or more of the following symptoms, will be found to coincide with the date of the accident; viz., shortness of breath on exertion, diminished power of endurance, fixed pain in the chest, back, or abdomen, or dry cough. In a few instances, after a period of incapacity for labour or exertion, but not characterized by definite symptoms, the injured persons have recovered ordinary health, and enjoyed complete exemption from suffering for long periods, after which symptoms of a definite character have been exhibited, and have continued with increasing severity, till the fatal termination of the disease.

The preceding observations may serve to explain the special

* *Vide Cases 135, 137, 138.*

† *Case 135, e. g.*

liability to aneurism of the aorta, which is notoriously connected with certain pursuits and handicrafts requiring great and continued muscular effort with distended lungs, or fatiguing exercise with a constricted chest. Porters, sledgers, soldiers, and bandsmen, exhibit a much larger percentage of aortic aneurism than others not similarly employed. Aneurism of the aorta is usually fatal; but the examples of temporary arrest or permanent cure of the disease, recorded within the last ten years, are sufficiently numerous to encourage the hope of still greater success in its treatment. These remarkable results are partly due to the more accurate knowledge now possessed by physicians of the conditions which favour, and those which retard or prevent coagulation of the contents of the sac, and likewise, in some degree, to the discovery of new and more potent agents for effecting that object; but they are mainly to be accredited to the greater boldness and skill with which the resources of surgery have been latterly employed in the treatment of aneurism.

Owing to the indefinite character of the symptoms which, in many cases, attend aneurism of the aorta in its early stages, and still more to its frequent latency, especially within the thorax, it is difficult to determine the average duration of the disease in a large number of cases. Where, however, as has repeatedly happened, symptoms indicative of injury to the aorta are associated with a definite strain or concussion of the chest or back, and followed by those of aneurism of that vessel, the date of commencement of the disease may be fixed with accuracy. In the cases recorded in Tables XVI. and XVII., I find the duration of aneurism of the aorta within the chest to have varied from ten days to ten years; and that of aneurism of the abdominal aorta to have ranged from fifteen days to eleven years.

The longest duration of aneurism of the aorta witnessed by Walshe was thirty-eight months. Dr. C. J. B. Williams has mentioned the case of a gentleman then living, who, to his knowledge, had been the subject of aneurism for thirty years.*

In general terms, the duration of aneurism of the aorta may be said to vary from a few weeks to as many years. It depends upon many circumstances, having reference to the constitution

* *British Medical Journal*, May 12th, 1870.

and the contents of the sac, the size, situation, and bearings of the tumor, the complications of the disease, and the previous health and present habits of the patient. A true aneurism of small size, so situate as not to interfere materially with important organs, in a person of previously unimpaired health, favourably circumstanced in regard to treatment, and submissive to restriction, offers the most favourable conditions under which the disease can be presented. A false aneurism, lined to a great depth by solid laminated fibrin, and circumstanced as above mentioned, affords a scarcely less favourable prospect. Conditions the reverse of the foregoing, and proportionately to their respective gravity, would suggest an unfavourable prognosis.

As to the effect of aneurism of the aorta upon the left ventricle of the heart, opposite opinions have been expressed by pathologists; some eminent writers having held that it necessarily leads to hypertrophy,* a view which, having reference to the impaired elasticity of the portion of the arterial wall constituting the sac, would seem theoretically to be well founded. Nevertheless, I am convinced that it is erroneous.

I believe that left ventricular hypertrophy exists in connexion with aneurism of the aorta, only where valvular disease or some other fully recognized cause of hypertrophy has been in operation.† In reference to this question, Tables XVI. and XVII. include 48 cases in which the condition of the valves, of the aorta, and of the left ventricle, is positively stated. Of this number, 8 exhibited disease of the aortic valves alone, or of the aortic and mitral valves conjointly; hypertrophy of the left ventricle existed in all these cases in connexion with aneurism of the aorta. Hypertrophy of the left ventricle, not associated with valvular disease, existed in 9 cases; but in 7 of these, extensive and advanced atheroma of the aorta coexisted with aneurism, and in 3 of the cases the hypertrophy was very slight. Atheroma of the aorta beyond the seat of aneurism was present in 20 cases in which hypertrophy of the left ventricle did not exist, and in 4 of these the left ventricle was in a state of atrophy, whilst, in

* Hope, *Diseases of the Heart and Great Vessels*, third edition, pp. 435 and 450, foot note.

† *Vide*, p. 488.

12 others in which hypertrophy did not exist, the aorta was apparently healthy, except at the seat of the aneurism. In 2 instances the left ventricle was dilated and fatty (not hypertrophied) in connexion with extensive atheroma of the aorta. Thus, excluding the cases in which left ventricular hypertrophy was accounted for by lesion of the valves, 9 cases only, or a fraction over one-fifth of the remainder, can be adduced in support of the alleged dependence of hypertrophy of the left ventricle upon aneurism of the aorta. But, in most of these cases, the patients were mechanics or soldiers, and therefore exposed, in their daily avocations, to causes adequate to the production of functional hypertrophy; and in 7 of them the aorta was extensively atheromatous, a condition in itself capable of producing hypertrophy of the left ventricle.* Finally, in 32 cases out of 48, aneurism of the aorta existed without hypertrophy of the left ventricle, and in 4 of these the ventricle was actually in a state of atrophy. Sir Dominic Corrigan has expressed a very decided opinion on this question. He says: "Aneurism of the arch of the aorta has no tendency to produce enlargement of the heart."† Dr. Stokes declares that "where the heart or its valves have not been previously engaged, there is no reason to believe that the existence of aneurism in any portion of the aorta throws additional labour on the heart, and hence, we commonly find a healthy heart coexisting with a vast aneurism."‡ Professor Axel Key has come to a similar conclusion.§ Walshe, however, believes that aneurism of the aorta, near the heart, is capable of producing hypertrophy of the left ventricle.||

The *diagnosis* of aneurism of the aorta has reference to, (a) its identity, (b) its variety, and (c) its precise seat of origin. The positive determination of these several points, as far as it can be accomplished, constitutes a necessary preliminary to prognosis and treatment. It is, however, in many cases attended with

* *Vide* Case 101, p. 889.

† *Proceedings of the Pathological Society of Dublin*, February 13th, 1841.

‡ *Diseases of the Heart and Aorta*, p. 579.

§ *Vide* p. 486 for a full discussion of this subject. Since the date at which this passage was written, I have treated three additional cases of aneurism, which are included in the foregoing summary.

|| *Diseases of the Heart and Great Vessels, opus citat.*

great, and in a few, with insuperable difficulty. As to the actual existence of aneurism in any given case, suspicion of its presence having been once aroused, a careful scrutiny of the evidence presented will very rarely fail to lead to a positive conclusion.

The *symptoms* or rational signs of aneurism of the aorta may be classified under the two heads of "subjective" and "objective;" whilst the "physical signs," by which is meant the evidence of aneurism, whether proper to the tumor itself, or due to its relations with other parts, discoverable by auscultation, percussion, and palpitation, may be divided accordingly into "intrinsic" and "extrinsic" signs. In practice, however, it will be found more convenient and more instructive to study the disease in its clinical aspect, as presenting definite groups of phenomena, to each of which is assigned a value, determined not only by its special and individual significance, but likewise by its association with other phenomena, whether symptoms or signs.

It will be convenient to discuss, in the abstract, the symptoms and signs which indicate the presence of aneurism in the chest and in the abdomen respectively, before proceeding to consider the modifications which they may present, according to the variety, the point of origin, and the position of the aneurism in both these situations.

The symptoms of aneurism are, for the most part, due to excentric pressure. These may arise, either from the direct pressure of the tumor upon adjacent organs and structures, or from the irritation of parts more or less distant, through the medium of sentient, motor, or vaso-motor nerves.

The pain of aneurism is mainly the result of excentric pressure upon adjacent sentient nerves, and is referred to the seat of their distribution. It is usually shooting, paroxysmal, and of extreme severity, not unfrequently eliciting cries of agony from the unhappy sufferer; it is likewise intermittent. The pain of abdominal aneurism, especially of that which is located behind the crura of the diaphragm, or implicating the solar plexus, is much more severe than that which attends the disease when located in the chest.

The consequences of the mechanical pressure of an aneurism

are not only various, according to the organs affected by it, but, owing to the remarkable proneness of aneurismal tumors to vary in direction and rate of growth, the symptoms arising from the pressure exercised by them upon particular organs and structures are singularly variable and remittent. It is necessary to consider the effects of excentric pressure in the thorax and in the abdomen, respectively.

Thoracic aneurism most frequently tends to the anterior surface of the chest. Hence, it may encroach upon the sternum, the ribs, costal cartilages and intercostal muscles, and the clavicles; absorbing or displacing in its progress such of these parts as it happens to bear upon, and ultimately projecting on the external surface in the form of a yielding and pulsating tumor. The symptoms which attend the advance of an aneurism to the front of the chest are limited to pain of a dull or aching character, extending, not unfrequently, to either shoulder, to the corresponding side of the neck and head, and down the arm, in the course of the cervical and brachial nerves. Severe darting pains of a neuralgic character, radiating round the chest in the course of the intercostal nerves, are likewise occasionally felt even before the tumor has appeared upon the surface. The advance of an aneurism of the lower portion of the arch, or the thoracic aorta, may compromise the left bronchus or lung; obstruction to the circulation of air, or disorganization of the pulmonary structure, being the result. The heart may be dislocated forwards by an aneurism situated low down in the thorax, whilst, by the lateral enlargement of such an aneurism, the œsophagus or the right bronchus may be obstructed or occluded. By the expansion of the sac of an aneurism implicating the anterior wall of the chest, collateral parts may be also pressed upon; hence, frequent obstruction of the descending cava, and venous congestion of the upper portion of the body; hence, likewise, implication of the pulmonary artery, or of either auricular appendix. The tumor may ascend into the neck, and by its pressure upon the innominate, the carotid, or the subclavian arteries, cause arrest, partial or complete, of arterial circulation in the parts supplied by these vessels, with its various consequences; or it may produce venous stasis by compressing the innominate, the internal jugular, or the subclavian veins. Irritation or paresis of the

several nerves at the root of the neck may likewise result from the upward growth of an aneurism of the arch of the aorta. Thus may be explained numbness or paralysis of the upper extremity on the side affected, dilatation or contraction of the pupil on one, or even on both sides, and the various symptoms having reference to the larynx, viz., dysphonia, stridor, aphonia, and spasm. The backward growth of an aneurism springing from the ascending or the transverse portion of the arch of the aorta, may occasion pressure upon the trachea, the right or left bronchus, the œsophagus, the thoracic duct, the sympathetic nerves, the spinal column, or the ribs; whilst an aneurism of the descending portion of the arch, or of the thoracic aorta may, by its growth in the same direction, compress the last mentioned parts only. Finally, an aneurism arising from the root of the aorta, and enlarging mainly in the direction downwards, may implicate any of the chambers of the heart.

Aneurism of the primary portion of the aorta, growing from one or more of the sinuses of Valsalva, has been most amply illustrated by Thurnam,* who has collected from various sources 18 cases of aneurism in this situation. In 2 of these the aneurism was double, and in 1 it was treble. Thus, there were in these cases 22 aneurisms, of which the right anterior sinus was the seat of the disease in 12 instances, the left anterior sinus in 4, and the posterior sinus in 6. Of the 22 aneurisms, no less than 13 had grown downwards, and projected or opened into the chambers of the heart. This tendency of aneurism connected with the primary portion of the aorta, had been previously noted by Professor Smith,† and by him attributed to the backward pressure exercised upon the column of blood in this portion of the vessel during the systole of the aorta.

The rule is not, however, without many exceptions. Of 7 examples of aneurism of the sinuses of Valsalva collected from the *Proceedings of the Pathological Society of Dublin*, for the eight years 1863-70, 4 had grown downwards, engaging one or other of the chambers of the heart, and 3 had grown upwards.

As regards the special tendency of aneurisms arising from particular sinuses, to establish a communication with definite por-

* *Medico-Chirurgical Transactions*, vol. xxiii., 1840, page 323.

† *Dublin Journal of Medical and Chemical Science*, vol. ix.

tions of the heart, Dr. Thurnam's conclusions possess some interest; he has found that those which take origin in the anterior and right sinus are the most liable to open into the right chambers, the pulmonary artery, and the pericardium; those of the anterior and left sinus have a special tendency to open into the left chambers, or the pulmonary artery; whilst aneurisms of the posterior sinus are most likely to communicate with either auricle, but especially with the right. Aneurisms arising from the ascending portion of the arch, above the sinuses, have a tendency to open into the pericardium, the descending cava, the pulmonary artery or its right branch, the right pulmonary veins, or either bronchus. According to Dr. Sibson's calculations these aneurisms have ruptured into the pericardium in the proportion of 45 per cent., into the pulmonary artery, 13·5 per cent.; into the right auricle, 8·5 per cent.; and into the right and left ventricle respectively, 5 per cent.*

Of the 68 cases given in Table XVI., the aneurism had arisen from the ascending portion of the arch in 30 instances, including an example of double aneurism. The direction of growth of the sac was forward in 9 instances, forward and to the right in 4, forward and to the left in 2, forward and upward in 1, forward upward and backward in 1, backward and to the right in 5 (in 4 of which the superior vena cava had been either compressed or opened into), upward and to both sides in 1, downward and to the right and opening into the right ventricle in 1; there was general expansion of the sac in 4 instances.

Innominate aneurism may, by its pressure upon adjacent parts, give rise to extrinsic phenomena identical with those caused by the upward growth of an aneurism of the ascending portion of the arch of the aorta, or of the first half of the transverse portion.

In the abdomen, aneurism of the aorta may, by its backward growth, erode the spinal column, and even penetrate the spinal canal. It may likewise, after perforating the diaphragm, cause absorption of the false ribs, and appear externally in the inferior dorsal region; or it may project on either side, usually the left, between the last rib and the crest of the ilium. By its growth in this direction, an aneurism of the abdominal aorta may like-

* *Medical Anatomy*, p. 53.

wise make pressure upon either kidney or ureter, the pœ muscles and lumbar nerves. If it advance towards the anterior wall of the abdomen, the pancreas, the liver or bile-ducts, the hollow viscera, or the solar plexus, may be implicated. By its lateral growth, an aneurism within the abdomen may compress or open into the inferior cava, and thereby cause venous stasis in the lower half of the body. It may ascend into the chest after penetrating the diaphragm, and implicate the pleura or lung on either side, or the pericardium. Finally, it may descend into either iliac fossa, or into the true pelvis, involving in its progress the organs and parts lodged in these recesses. The pain caused by the pressure of an abdominal aneurism upon the solar plexus is intermittent, paroxysmal, and of the most intense severity; that connected with the process of gradual absorption of the vertebræ, is dull, persistent, and boring in character, as shown by Dr. Law.*

The symptoms of thoracic aneurism, although in exceptional instances liable to variation in regard to locality, intensity, and combination, possess a definite value, and, once clearly identified, afford a standpoint whence, by a careful process of exclusion, a positive diagnosis may in most instances be arrived at. But, owing to the rigid character of the thoracic parietes, the great capacity of the chest, and the variable volume of the lungs, the presence of an intra-thoracic tumor, even of considerable magnitude, is not always announced by definite symptoms; and when those which indicate the presence of a tumor are well pronounced, it becomes necessary to determine, in the first instance, whether it is or is not aneurismal.

The symptoms indicative of aneurism within the chest may be now considered in detail. They include pain (intrinsic and extrinsic) visible or palpable tumor and the effects of pressure upon (a) blood vessels, (b) the laryngeal nerves, (c) the trachea, bronchial tubes, or lungs, (d) the cesophagus, (e) the sympathetic or the cardiac nerves, (f) the heart, (g) the thoracic duct, (h) the diaphragm, and (i) the vertebræ.

The intrinsic pain of thoracic aneurism, though not urgent, is not the least significant of its symptoms. It is localized, deep-seated, and constant, oppressive and constrictive rather than sharp, and readily aggravated by causes which quicken the cir-

* *Dublin Journal of Medical Science*, vols. xxi. and xxii.

culation, and is then associated with dyspnoea in greater or less degree. This pain is caused by tension and subacute inflammation of the sac, and by pressure upon its nerves of supply; hence, the alleviation of it which follows the abstraction of blood or other measures calculated to reduce intra-vascular pressure.

Extrinsic pain, or that due to pressure upon adjacent sentient nerves, is of a totally different character; it is sharp and paroxysmal, ill-defined, wandering, and eminently neuralgic in character; it is usually most severe at night, and, because existing in parts very rarely invaded by neuralgia or muscular rheumatism, namely, the superior dorsal region, neck, walls of chest, and either arm, is of the utmost diagnostic value. The parts affected by these pains may be either benumbed or hyperæsthetic. Where other symptoms suggestive of aneurism likewise exist, the significance of flying pains of this character in the situations mentioned will be proportionately enhanced; but occasionally they constitute the only actual symptom of aneurism,* and should never be regarded with indifference. The patient speaks of those pains as "rheumatic," and generally connects them with casual exposure to cold or the use of damp clothing, but they are uninfluenced by atmospheric changes, and by anti-rheumatic treatment.

The existence of a pulsating tumor in the course of the aorta at any point of the surface of the chest, but especially on its anterior or posterior aspect, or projecting into the neck, though not amounting to absolute proof, affords stronger evidence of aneurism than any other isolated symptom. The characteristics of an aneurismal tumor are of two kinds; namely, those which belong to it in its simplest form, and those which are incidental to it. The former are: softness, combined with tension and elasticity; decrease of volume under pressure; a strong, heaving, and expansile impulse synchronous with that of the heart; and progressive growth. One or more, or even all, of these features may be replaced by accidental changes in the sac, or its contents. Thus, the tumor may have become, in greater or less degree, solid by deposit of fibrin or coagulation of blood within the sac. In such case the qualities of softness and elasticity, yielding un-

* See Case 139.

der pressure, and systolic expansion of the aneurism, might be greatly modified, or even entirely abolished, and the growth of the tumor arrested.

It is further characteristic of an aneurismal tumor appearing upon the exterior of the body, and taking the ordinary course, that, at an advanced stage of its progress, it exhibits upon its surface one or more projections, which are red, tender, and more yielding than the surrounding portions. Here the sac has been thinned or perforated by ulcerative absorption, and its tegumentary or mucous investment is in progress towards sloughing. The retrocession or disappearance of a tumor, previously visible and palpable, whilst common to all those with liquid contents, is characteristic of aneurism as contrasted with cancerous growths, and coincides with the enlargement of the sac at some other point or points of its surface. I cannot acquiesce in the opinion expressed by Dr. Walshe, that cancerous tumors of the mediastinum, no less than those which are aneurismal, may recede within the chest after having appeared externally.* Dr. Walshe has not adduced an example of such a phenomenon, nor has Dr. Risdon Bennett in his exhaustive treatise.† The appearance of a tumor on the surface of the chest in the course of the aorta, should not only raise a suspicion of aneurism and suggest a careful examination with a view to diagnosis, but the existence of an aneurism having been positively determined, the situation of the tumor may serve to indicate, with more or less precision, the portion of the aorta whence it takes origin.

A tumor unmistakably aneurismal, situate upon the right side of the chest, between the first intercostal space and the third costal cartilage, and inside the nipple-line, would, most probably, be connected with the ascending portion of the arch above the sinuses of Valsalva. Aneurism arising from one or more of the sinuses usually *descends*, and would, therefore, present on the surface, if at all, below the level indicated; whilst innominate aneurisms usually project above that level, and *ascend* into the neck. It is, however, necessary to bear in mind that exceptions to both these statements, especially the former, are occasionally met with. The preponderating frequency of aneurism in this portion of the aorta, as compared with all other portions of it,

* *Diseases of the Heart*, 4th edition, 1873. † *Intra Thoracic Growths*, 1864.

and with the *arteria innominata*, should be duly estimated where doubt exists. An aneurism centred at the right margin of the sternum, the inner portion of the second costal cartilage, and first interspace, has its origin most probably at the first curve of the arch, implicating, or not, the root of the innominate artery; a point to be determined by reference to the circulation in the right carotid and subclavian arteries.

Aneurisms of the *innominata* rarely point at this level, and still more rarely fail to appear, at an early stage, above the clavicle. The downward extension of percussion-dulness, and the diffusion of impulse in the same direction, would, in such a case, be in favour of the diagnosis of aortic, and against that of innominate aneurism.

A pulsating tumor, having its point of greatest prominence at the centre of the *manubrium sterni*, especially if an impulse were perceptible above the fourchette, and venous congestion existed at the root of the neck, would indicate aneurism of the transverse portion of the arch somewhere about its centre; whilst aneurism of the left extremity of this portion of the vessel would be most likely to point beneath the left clavicle. In a case observed by Dr. Mayne, aneurism of the ascending portion of the arch had pointed to the left of the sternum, making pressure upon the left bronchus;* and in another, recorded by Dr. Greene, an aneurism of the descending aorta had passed over to the right side behind the *œsophagus*, and compressed the right bronchus.†

Aneurisms of the descending portion of the arch, or of the thoracic aorta, pointing externally, most frequently appear in the left scapular, or infra-scapular region; they may, however, project above the left clavicle.

Diminished force, or even total suppression of circulation in adjacent arteries, or in those arising from the sac, may be caused by the excentric pressure of an aneurism of the aorta, by the formation of an autochthonous clot, the impaction of one detached from the sac, or by the extension of the contained coagula

* *Proceedings of the Pathological Society*, December 20th, 1851; see also a case by Dr. T. Evelyn Little, *ibid.*, vol. v., part ii.

† *Dublin Journal*, vol. vii., 1835.

over the mouth of the vessel. Between these several causes of obstructed arterial circulation, the presence or absence of a pulsating tumor, and its position with regard to the obstructed vessels, must serve as the principal means of distinction. Where no tumor exists, the probability of endarteritis with plugging might be inferred. Mahomed declares that the pulse-tracings exhibit no special characters distinctive of obstruction from these several causes.*

I have observed postponement of the radial pulse, first noticed by Henderson, as a sign of aneurism of the arch, in one case (146) only.

Partial suppression of pulse in the carotid or subclavian artery, or in both these vessels, on one side of the body, may be likewise due to the pressure of a mass of enlarged lymphatic glands in the anterior mediastinum or in the neck.† In connexion with thoracic aneurism, these phenomena are not unfrequently exhibited in the carotid or subclavian artery of either side, and occasionally in both these vessels on one side of the body. Complete occlusion of the common carotid and vertebral artery on one side, has been followed by hemiplegia from anæmia and atrophy of that side of the brain. The existence of obstruction in the vessels of *both* sides is characteristic rather of cancerous tumor than of aneurism. Inequality of radial pulsation may be readily detected by the finger, but the crucial test is supplied by the use of the sphygmograph. It is noteworthy that, in arteries obstructed by an aneurism, whilst the usual progress leads gradually to permanent suppression of pulsation, the pulse may exhibit considerable variations of volume within a brief period; it may be even temporarily suspended, and, by removal of external pressure from the vessel, subsequently restored to its normal condition.‡ Such variation in the pulse, where attributable to a tumor, is eminently suggestive of aneurism.

The pressure of an aneurism upon the great veins within the chest is indicated by venous turgescence, with œdema, of the portions of the body whence blood is ordinarily returned to the heart through the compressed vein. Congestion and œdema of the right side of the head and neck only, with turgescence of

* *Medical Times and Gazette*, August 30th, 1873.

† *Vide* Case 71, p. 736.

‡ *See* Case 142.

the external jugular vein, would indicate pressure upon the corresponding internal jugular, and if the compressing body were an aneurism, its seat would most probably be the innominate or the common carotid artery. On the left side, congestion similarly limited would indicate the pressure of a carotid aneurism. Localized congestion and œdema of one upper extremity from the pressure of an aneurism, would point to the subclavian vein as the subject of compression and the subclavian artery as the seat of the disease. Simultaneous congestion of all these parts on one side would indicate pressure upon the corresponding innominate vein, from an aneurism of the transverse portion of the arch, if on the left side, and of the innominate artery on the right. Congestion and œdema, limited to the upper half of the body, and depending upon the pressure of an aneurism, would indicate the ascending portion of the arch, or the right extremity of its transverse portion, as the seat of the disease.

The symptoms produced by the pressure of an aneurism upon the trachea, the bronchi, the lungs, or the recurrent nerves, are remarkably various, and of the utmost diagnostic value. It occasionally happens that, with the exception of the symptoms so produced, no others decidedly suggestive of tumor within the chest, can be detected.* Yet, so characteristic are they, that, when duly estimated, they constitute in themselves evidence sufficient to warrant a positive diagnosis of intra-thoracic tumor, the nature of which must be determined by collateral symptoms and signs.

The symptoms referable to the larynx may be first considered. These are the result either of spasm or paralysis of the intrinsic muscles of the larynx, affecting one side only, or both sides, from the pressure of an aneurism upon the pneumogastric or the recurrent nerves. Spasm of the larynx is most frequently unilateral, and a consequence of partial pressure upon the recurrent laryngeal nerve of the same side; it is, however, very rarely unaccompanied by the symptoms of tracheal pressure. It is characterized by paroxysms of the most urgent dyspnoea, accompanied by laryngeal stridor, and usually brought on by exertion or by excitement of any kind. During the paroxysm, the features are haggard and expressive of great alarm, the pupils are

* *Vide Case 138*; also a case reported by Drs. O'Ferrall and Stokes, in the *Proceedings of the Pathological Society of Dublin*, March 24th, 1860.

dilated, the face more or less congested and of a leaden hue, the nostrils are dilated, and the muscles of the chest and shoulders in a state of tension during the struggle for breath. The action of the heart is tumultuous, the carotid and subclavian arteries bound visibly, whilst the radial pulse is quick, small, and hard. The agony may last for several hours. It not unfrequently terminates in syncope, from which the patient gradually recovers, and is then quite free from suffering till the occurrence of the next paroxysm; or death may be the result of one of these seizures.* During the interval, which is of uncertain duration, the voice is unaffected, and there may be a total exemption from symptoms; but, usually, there is more or less difficulty of breathing. Respiration is readily quickened by exercise, and then the patient has harsh and dry cough of a sharp and metallic character. As pointed out by Dr. George Johnson, the stridor is most distinctly audible, during a paroxysm, over the middle cervical vertebræ,† and laryngoscopic inspection during the intervals will show that the larynx is in its normal condition.‡

More decided pressure upon the pneumogastric or recurrent nerve of either side, will cause paralysis of the laryngeal muscles. This is usually limited to the side affected; but in two cases reported, one by Dr. Bäumlér,§ and another by Dr. Johnson,|| bilateral paralysis of the larynx resulted from pressure upon the pneumogastric and recurrent nerves of one side only.¶ When laryngeal symptoms are the result of paralysis, there is either aphonia or permanent huskiness of voice, and if an exa-

* Vide the case of the late Earl St. Maur, by Dr. C. J. B. Williams, pamphlet, 1870.

† *British Medical Journal*, May 23rd, 1874.

‡ See case 138.

§ *Transactions of the Pathological Society of London*, vol. xxiii., p. 66.

|| *Ibid.* vol. xxiv., p. 42.

¶ In a more recent communication (*British Medical Journal*, December 19th, 1874), Dr. Johnson has expressed the opinion, that in these and all similar cases, paralysis of the laryngeal muscles of the opposite side is due to a reflex influence conducted to the opposite recurrent nerve, through the commissural connexion between the spinal accessory nuclei. I cannot conceive how reflex paralysis of the opposite recurrent nerve, which must be necessarily *partial*, as influencing only the fibres of origin on the side of pressure, could produce *complete* paralysis of the muscles supplied by it. No such difficulty exists in regard to the theory of reflex spasm.

mination be made with the laryngoscope, the arytenoid cartilage and vocal chord of one side, or those of both sides, will be found immovable during forced inspiration and the attempt to vocalize, whilst the larynx is obviously free from congestion and the other usual causes of hoarseness. As in ordinary cases of paralysis, the muscles of the larynx, if long paralysed, become wasted. Dr. Morell Mackenzie has published two cases of aneurism of the arch of the aorta, causing paralysis of the muscles of the left side of the larynx by pressure upon the left recurrent nerve. In one of these cases, which was of eighteen months' duration, the paralysed muscles had undergone atrophy.* Aneurisms which give rise to laryngeal symptoms by pressure upon the pneumogastric or recurrent nerves exclusively, most frequently arise from the upper extremity of the ascending, or the transverse portion of the arch, and from the anterior or the superior surface of the vessel; hence, they are announced by visible tumor, perceptible pulsation, venous congestion in the neck, and other palpable evidence of their existence. The affected nerves are always upon the stretch by the expansion of the sac, and not unfrequently they are flattened upon, and incorporated with it.

Pressure upon the trachea may result from an aneurism arising from any portion of the arch of the aorta, or from the arteria innominata. When produced by an aneurism of the ascending portion of the arch, or by one connected with the anterior or the superior surface of its transverse portion, the symptoms of pressure upon the trachea are *late* in the order of events, and are usually associated with those of irritation or paralysis of the laryngeal nerves, and with dysphagia. Such is likewise the case in regard to aneurisms of the descending portion of the vessel. The most urgent symptoms of tracheal pressure are, however, in many cases, produced by an aneurism springing from the posterior surface of the transverse portion of the arch. In such connexion, they are, moreover, of early occurrence in the history of the case, and sometimes constitute the first, and, for a long period, the only symptom of the disease. Although the symptoms of tracheal pressure constitute the most salient feature in many cases of aneurism, they are, in my opinion, very

* *British Medical Journal*, May 21st, 1870.

rarely unassociated with those of laryngeal spasm, and both may be produced by an aneurism not implicating the recurrent nerves.* The tracheal and bronchial nerves can scarcely escape being involved, and reflex irritation through these nerve-filaments, intimately associated as they are with the pneumogastric and its branches, will account for the usual association of laryngeal symptoms with those of tracheal pressure.

The trachea, when pressed upon by an aneurism, is flattened and more or less indented upon one side, and, when the pressure is extreme, stretched and bent towards the opposite side. When the trachea is laid open, its lateral wall is seen to bulge inwards at the point of pressure, and, if the cartilages have been already eroded, the mucous membrane in that situation is congested, of a deep purple colour, or perhaps in a state of incipient gangrene preparatory to irruption of blood from the sac. Owing to the peculiar conformation of the trachea, and the laxity of its connexions, it readily eludes pressure from the front, moving to either side according to the inclination of the impinging body, and receiving the impression upon the side opposite. The trachea, when compressed by an aneurism arising from the ascending portion of the arch, from the right extremity of its transverse portion, or from the arteria innominata, receives the pressure upon its right surface; but when the aneurism arises from the left portion of the arch, whether transverse or descending, the trachea, if encroached upon by the sac, will receive the pressure upon its left side.

Owing to the relative positions of the trachea and the arch of the aorta, whence compressing aneurisms most frequently spring, the portion of the former usually pressed upon by an aneurism is that immediately above the bifurcation. The symptoms and signs which indicate pressure upon the trachea, not implicating the larynx, are the following: Dyspnoea, with a feeling of tightness and obstruction behind the upper portion of the sternum; stridulous inspiration, the stridor being loudest over the superior dorsal spine, as indicated by Dr. Johnson, or "from below," as long since noted by Dr. Stokes; a dry and harsh cough, with a decided metallic clangour; and voice of a muffled or distant and ventriloquial character. These phenomena become more significant if intensified by exertion or excitement. Facial

* See Case 138.

decubitus, and frequent extension of the neck, have been noted by Dr. Bellingham and Dr. Law, respectively. Examination by means of the laryngoscope will show that the larynx is unaffected, the glottis expanding fully with inspiration, and voice of the character above mentioned being readily educed. There is, in most cases, some inequality of respiration on the opposite sides of the chest, the vesicular murmur being less distinct on the side corresponding to that of pressure, and associated with tracheal breathing and voice at the upper part of the sternum and in the interscapular space on the same side. This is usually the result of pressure upon the main bronchus, but it may be caused by pressure upon the trachea exclusively. A tumor projecting into the trachea close to the orifice of either bronchus, may divert the current of air in some degree to the opposite side, and thus become the cause of unequal inflation of the lungs; whilst the implication of the bronchial nerves in the sac may excite spasm, and lead to the same result. In Case 138 the inequality of respiration would seem to be explicable only on one or both of the foregoing hypotheses. Suppression or decided feebleness of respiration in one lung, if persistent, would indicate mechanical obstruction of the bronchus of that side; and, if associated with the vocal and other phenomena above mentioned, engagement of the trachea might be likewise inferred. Aphonia cannot be included amongst the ordinary symptoms of tracheal or bronchial compression; in one case, however, observed by Professor Smith, and referred to by Dr. Stokes,* in which the trachea alone was implicated, aphonia existed without stridor.

Where the symptoms of laryngeal spasm are superadded to those of tracheal compression, as they usually are towards the termination of every case in which the trachea is implicated by an aneurism, the most urgent respiratory distress, not unfrequently ending in death by asphyxia, is exhibited. The larynx is rarely altered in structure, but in one such case (No. 142) well marked œdema of the glottis existed, most probably the result of venous congestion from pressure.

The symptoms produced by the pressure of an aneurism upon one of the bronchial tubes, without implicating the trachea, are few and indefinite previous to irruption of blood from the sac;

* *Opus citat.*

not so, however, the signs discoverable by auscultation and percussion. The left bronchus is more frequently the subject of pressure than the right, owing to its anatomical relationship to the descending portion of the arch and the thoracic aorta. The greater number of aneurisms within the chest tend to advance towards its anterior wall, and those connected with the ascending and transverse portions of the arch, which, by their backward growth, involve the right bronchus, generally implicate the trachea also. Hence, isolated bronchial pressure is most frequently witnessed on the left side. There may be defective expansion of the affected side of the chest, owing to imperfect inflation of the lung supplied with air by the obstructed bronchus, but so many causes, having reference to actual and antecedent conditions of the lung, may give rise to this symptom, that, apart from other evidence, it must be regarded as of little practical value for the purpose of diagnosis. The degree in which it is exhibited where obstruction of a bronchus, not amounting to complete occlusion, exists, is, moreover, very trivial, and by no means readily detected. The method proposed by Sir Dominic Corrigan,* whether for the purpose of detecting imperfect expansion or deep-seated pulsation at any point of the surface of the chest, is by far the most delicate and trustworthy. This consists in placing the patient in a strong light, the chest having been entirely denuded, and then looking downwards obliquely from behind the shoulder on the affected side.

Complete occlusion of a bronchus by the pressure of an aneurism is exceedingly rare. Dr. Stokes declares he has never met with an example of it.

Cough is another and a constant symptom of bronchial pressure. It is generally associated with bronchitis, and hence, regarded separately, it avails very little as evidence of aneurism or other tumor within the chest. Owing to pressure upon the pulmonary plexus the cough may partake of the ringing or aneurismal character, and when this is the case, it possesses a decided and positive significance. Occasionally the mucus expectorated is streaked with blood, and there may be veritable hæmoptysis from local congestion, or from leakage of the aneurism.

Doctor Greene† has observed retraction of the trachea with

* *Lancet*, February 7th, 1829. † *Dublin Journal of Medical Science*, vol. vii.

inspiration, in two cases of aneurism pressing on the left bronchus. Where the primary bronchus of either side is compressed by a tumor, the respiratory murmur and the vocal fremitus are proportionately diminished in the lung supplied by it; whilst in the opposite lung these phenomena are increased in an inverse ratio. Occasionally, also, as noticed by Dr. Greene and Dr. Stokes, air is heard to enter the affected bronchus "with a rush" at the acme of a forced inspiration. Inequality of respiratory sound is frequently noticeable, as between the upper and the lower portions of the lung affected by pressure upon its main bronchus. Respiration is, in such cases, usually more feeble in the apex than in the base of the lung, a difference which I believe to be due to pressure also upon the secondary bronchus through which the apex of the lung is inflated.* With defect of respiratory sound in either lung, are usually associated bronchial respiration and bronchophony in the scapular region, or the interscapular space of that side, whilst vocal thrill is imperfect, and may even be abolished, over those portions of the lung in which respiration is most enfeebled. With the signs above mentioned a clear sound on percussion is associated, where consolidation of the lung or pleuritic effusion does not exist. Amongst the consequences of bronchial obstruction by pressure *ab extra*, congestive pneumonia and gangrene of the lung must be included. Pneumonia of a low or asthenic character, and readily passing into gangrene, is a frequent result of the pressure of an aneurism upon the bronchial vessels and nerves, as shown by Carswell.† It is usually localized, and where considerable impediment to the entrance of air into the affected portion of the lung, by pressure upon the bronchus, also exists, it is distinguished from all other forms of pneumonia by the absence of vocal fremitus.‡ Pneumonia, characterized by the ordinary signs, may, however, result from the implication of the pulmonary structure by the direct pressure of an aneurism. In the progress of such cases, repeated hæmoptysis from leakage of the aneurism, or from rupture of the pulmonary capillaries, is a usual symptom.§

* *Vide Cases 141 and 142.* † *Pathological Anatomy.* ‡ *Vide Case 136.*

§ See two cases by Dr. Mayne, *Proceedings of the Pathological Society of Dublin*, April 6th, 1861, and January 4th, 1863.

Feeble respiration with percussion-resonance on one side of the chest may, however, be due to the following causes, irrespectively of aneurism: local thickening of the bronchial membrane and consequent obstruction, as pointed out by Andral;* local emphysema; the impaction of a foreign body in the bronchus, a cancerous tumor, or enlarged bronchial glands pressing on the bronchus. Dr. Greene has shown that not only feeble respiration in one lung, but dyspnoea, dysphonia, stridulous breathing, and violent spasmodic and ringing cough, may result from the pressure of enlarged bronchial glands upon one of the bronchi.†

Doctor Stokes has published a case in which all the foregoing symptoms, except dysphonia, together with dysphagia and most of the physical signs of aneurism, were produced by a large cancerous tumor occupying the posterior mediastinum.‡

The distinction between these several causes of unequal respiration on the opposite sides of the chest, must rest upon collateral evidence. Local bronchitis is exceedingly rare; indeed, it is characteristic of the bronchial membrane that the changes induced by local irritation are rapidly diffused over its entire tract. The detection of local emphysema by means of its proper signs cannot be attended with much difficulty; the prolonged expiratory wheeze and exaggerated percussion-resonance with which it is accompanied, will suffice to distinguish it.

The diagnosis of bronchial pressure caused by a cancerous tumor is often extremely difficult, and sometimes impossible. When growths exhibiting the characters of scirrhus or encephaloid cancer exist anywhere upon the exterior of the body, or in situations where their nature can be determined, a presumption in favour of internal cancer, and against the presence of aneurism, would be warranted. The persistency of pressure would be in favour of the same view, whilst the physical signs peculiar to both these diseases, to be discussed presently, would afford still more important aid towards the distinction. In the case by Dr. Stokes, above referred to, a single systolic murmur existed. This sign, in connexion with a tumor in the posterior mediastinum, was suggestive of cancer rather than of aneurism.

* *Clinical Medicine*, vol. i., Observation ii.

† *Dublin Journal of Medical Science*, vol. x., 1837. ‡ *Ibid.*, vol. xxi.

The obstruction resulting from the lodgment of a foreign body in one of the bronchial tubes is usually more complete than that produced by the pressure of an aneurism. Hæmoptysis and local inflammation are quickly induced by its presence, and, contrary to what obtains where aneurism is the cause of obstruction, respiration is *equally diminished over the entire lung*. Finally, the history of a misadventure in which a foreign body *may* have found entrance into the air passages, and the absence, previous to such occurrence, of all symptoms pointing to aneurism, would be, in themselves, almost conclusive as to the cause of obstruction.

Dysphagia is a very common symptom of thoracic aneurism. It may be due to mechanical obstruction from the direct pressure of the tumor upon the œsophagus, which is the ordinary mode of its occurrence; to spasm of the gullet from direct or reflex irritation of the œsophageal plexus; or to paralysis of the muscular wall of the œsophagus from destruction of its motor nerves, or from excessive dilatation.

Doctor Davy, of Terenure, has recently published a most instructive case of dysphagia from dilatation of the œsophagus, in which, in addition, many of the more specific symptoms of aneurism were exhibited; *e.g.*, epigastric pulsation with tenderness and percussion-dulness, pains in the back and shoulder, and a "tearing or raking" sensation at the epigastrium on attempting to swallow. Deglutition was, moreover, less difficult whilst he lay on the right side; and all these symptoms gradually followed a strain in lifting a weight ten years previously. There was no hæmatemesis or enlargement of the external glands. Death was ultimately caused by inanition, and, on examining the body, the œsophagus was found to be enormously dilated, measuring nine inches in circumference some distance from its termination, and eight inches above and below this point; it was capable of containing forty ounces of liquid. The muscular coat was hypertrophied, and, in the most dilated portion, the mucous membrane presented several recent ulcers, and a still greater number of cicatrices. The œsophagus presented a twist from right to left, where it passed through the diaphragm. This was, no doubt, the cause of dilatation and all the subsequent symptoms. The stomach was dilated and its walls were thinned; the pylorus was

healthy.* In this case it would have been impossible during life to affirm that aneurism did not exist.

Aneurisms of the transverse and descending portion of the arch, and of the thoracic aorta, are those which most frequently make pressure upon the œsophagus. An aneurism of the ascending aorta may, however, produce this effect. The late Dr. Eames presented to the Pathological Society of Dublin an example of the latter kind. Three aneurisms were connected with the ascending portion of the arch of the aorta, and of these, the lower arose from the posterior sinus of Valsalva, and, passing backwards, pressed upon the œsophagus, and caused dysphagia with boring pain in the back.† Dysphagia resulting from the direct pressure of an aneurism is usually remittent; it may even entirely cease, and return after a long interval.‡ It may likewise vary in site, as shown by Dr. Law,§ and, as noted by the same observer, it may exist in the recumbent posture only. The patient can, in most cases, swallow liquids with facility, but deglutition of solids is attended with pain referred to the upper, middle, or lower portion of the sternum, according to the seat of obstruction. The difficulty of swallowing has been occasionally overcome by varying the posture of the body whilst making the effort, a circumstance which might be regarded as conclusive evidence of obstruction of the œsophagus either by the pressure of a tumor, the nature of which must be determined by reference to collateral symptoms, or by torsion of the tube itself, as in the case given by Dr. Davy.

Simple pressure upon the œsophagus, in the absence of a resisting point of counter pressure, ingrowth of the sac, or irritation of the muscular tunics or motor nerves by local inflammation, is not attended with dysphagia. Hence, displacement of the œsophagus to either side is not necessarily followed by symptoms of obstructed deglutition. Owing to the laxity of its connexions, the yielding character of its structures, and its great elasticity, the œsophagus may undergo considerable displace-

* *Irish Hospital Gazette*, May 1st, 1875.

† Meeting of March 5th, 1870.

‡ *Vide Stokes, Diseases of the Heart and Aorta*, Case 72, p. 576.

§ *Proceedings of Pathological Society*, February, 1841.

ment into either pleural cavity, under the pressure of a smooth-surfaced tumor, without suffering impairment of function.*

Dysphagia from spasm is most frequently referred to the scrobiculus cordis. It is characterised, as spasm usually is, by suddenness of development and cessation, and by being provoked by *dry* rather than by *moist* solids. A feeling of sharp pain in a particular part of the gullet, at the moment of passage of the alimentary bolus at that point, in a case of dysphagia from the pressure of an aneurism, would indicate erosion of the mucous membrane and early communication with the sac by ulceration or sloughing.

Contraction of the pupil from paralysis of the cervical sympathetic was first observed by Pourfour du Petit, who likewise noticed, in connexion therewith, drooping of the upper lid, and œdema of the eye. Paralysis was induced by section of the nerve, and the observation, though made in 1712, was not published till 1727.† Dupuy, early in the present century, noticed the same results from division of the sympathetic nerve; but he further observed increased temperature of the ears, perspiration, and eruptions on the skin.‡ Dr. John Reid was the first who showed that contraction might result from pressure on the sympathetic.§ Biffi, on the other hand, noticed dilatation of the pupil as a result of galvanizing the upper end of the divided nerve.|| Bernard¶ and Brown-Séquard** confirmed the observations of Dupuy, and further extended our knowledge, by proving experimentally that, not only alterations of the pupil, but likewise decided modifications of vascularity, temperature, and sensibility, may result from irritation or paralysis of the sympathetic in the neck. About the same time Valentin pointed out that the nerve endowment of the iris was twofold, its radiating fibres or dilator muscle receiving filaments from the

* *Vide* Case 140, and Fig. lxxiv.

† *Mem. de l'Acad. des Sc. de Paris*, 1727, quoted by Longet, *Traité de Physiologie*, 3^{me} edit., 1869, tom. ii., p. 932.

‡ *Jour. de Méd. Chirurg. et Pharm.*, Décembre, 1816.

§ *Edinburgh Medical and Surgical Journal*, January, 1838, and August, 1839.

|| *Annali Universali di Medicina*, 1845, Longet. *Ibid.*

¶ *Comptes Rendus de l'Académie des Sciences*, Mars, 1852.

** *Mém. lu à l'Acad. des Sciences*, January, 1854.

sympathetic, and the circular fibres or sphincter, from the oculomotor nerve. He also showed that the ciliary fibres of the sympathetic were derived, primarily, from the spinal cord.* Budge and Waller about this date also, proved by a series of experiments, that the sympathetic nerve-filaments of the iris take origin from the portion of the spinal cord between the sixth cervical and the fourth dorsal vertebræ, which portion they accordingly proposed to designate as the "cilio-spinal" region.†

In 1838, Dr. Hare made what may be regarded as the first clinical observation bearing on this subject. He noticed contraction of the pupil in connexion with pressure upon the sympathetic from a scirrhus tumor in the neck,‡ and in 1850, Dr. McDonnell, of Montreal, observed both contraction of the pupils and ptosis from the pressure of a malignant tumor in the same situation.§ The first case in which contraction of the pupil was observed from the pressure of an aneurism was one published by Dr. Walshe.|| Dr. Gairdner's cases were next in order of time,¶ and, in the following year, a case in which perspiration, limited to the side of the face on which the pupil was contracted, was published by the same observer.**

In the same year in which Dr. Gairdner's memoir appeared, Dr. Banks recorded an example of a similar kind.†† In this case, in addition to contraction of the pupil, partial ptosis existed. It is unnecessary to pursue further the history of this remarkable discovery. Since the last mentioned date, the examples of derangement of the pupil from the pressure of an aneurism upon the sympathetic chord in the neck, recorded in the annals of medicine, have been very numerous, Dr. J. W. Ogle has collected from various sources twelve such cases.‡‡ I have myself observed several cases of the kind. The pupil is

* *De Functionibus Nervorum Cerebraliū*, Bernæ, 1852.

† *Vierordt's Archiv. für Physiol. Heilkunde*, 1852.

‡ *Medical Gazette*, 1838.

§ Quoted by Gairdner from *The Montreal Medical Chronicle* for 1858.

|| *Diseases of the Lungs, Heart, and Aorta*, second edition, 1853.

¶ *Edinburgh Monthly Journal*, August, 1855.

** *Ibid.*, 1856.

†† *Dublin Hospital Gazette*, vol. ii., 1855.

‡‡ *Medico-Chirurgical Transactions*, vol. xli.

not insensible to light; it varies in diameter according to the degree of exposure; but it is always less affected by light or shadow than the pupil of the other eye, nor is sight in any degree impaired. For many of the facts and references in the foregoing *resumé*, I am indebted to the learned author of the exhaustive memoir last referred to (Dr. Ogle).

Constriction of the pupil on both sides may result from bilateral compression of the sympathetic. Of this phenomenon, which is exceedingly rare, an example has been observed by Dr. Gairdner, as a consequence of the extension of a large aneurism of the descending and thoracic aorta to both sides of the vertebral column.

As might have been anticipated from the recorded results of section of the sympathetic in animals, elevation of temperature, local perspiration, ptosis, and internal strabismus, have been repeatedly observed in connexion with contracted pupil from pressure on the sympathetic. The first and the third phenomena were exhibited in Case 142. Dilatation of the blood-vessels from paralysis of the vaso-motor nerves will serve to explain the rise of temperature and sweating. Ptosis and convergent squint, the latter of less frequent occurrence than the former, which is always incomplete, may, as suggested by Dr. Ogle,* be rationally referred to the double innervation of the levator palpebræ and abductor oculi muscles, through the filaments of communication from the sympathetic to the third and sixth cerebral nerves in the cavernous sinus. One of these two sources of nerve-supply being cut off by paralysis of the sympathetic, the muscles named lose their involuntary functions, whilst remaining under the control of the will. Hence, drooping of the upper lid, and arrest of nictitation, with continued power of opening the eye by a voluntary effort. Dilatation of the pupil has been likewise, but less frequently, observed in connexion with pressure upon the sympathetic; it arises from *irritation* of the nerve, and therefore indicates a less degree of pressure. Dr. Ogle has recorded 7 examples of this phenomenon, viz., 3 as the result of aneurism, 2 from scirrhus growths, one from the pressure of enlarged lymphatic glands,

* *Loco citat.*

and one from ecchymosis of the cervical portion of the spinal cord. In the two last mentioned cases, both pupils were dilated. Contraction has been observed to supervene upon dilatation of the pupil, and, in one case, to alternate with it.* Both contraction and dilatation of the pupil, with the attendant phenomena above mentioned, have been likewise observed in connexion with injury of the cilio-spinal region of the cord.†

Modifications of the pupil, in connexion with intra-thoracic or cervical tumor, furnish evidence of nerve-pressure only. The value of this symptom, as a diagnostic of aneurism, is rather relative than absolute, and must be determined by collateral evidence. Associated with any of the symptoms or signs of aneurism, changes of the pupil possess a positive value, not only as contributory evidence of the existence of that disease, but as indicating its locality, and in some degree, the dimensions of the sac.

Doctor Gairdner believes that symptoms of an anginal character may result from pressure upon the cardiac plexus by an aneurism close to the heart.‡ Such symptoms, however, apart from those of paroxysmal dyspnoea from laryngeal and bronchial spasm, which are necessarily accompanied by cardiac excitement, are very rare in connexion with aneurism. Another symptom, however, of kindred character, is occasionally witnessed in cases of aneurism of the aorta, namely, rhythmical irregularity of breathing, or "Cheyne Stokes' respiration."§ I have already, at p. 626, discussed this remarkable phenomenon, and at p. 632, propounded, with diffidence, a theory of its development.

Von Dusch, in 1867, declared that he had observed this symptom in certain forms of cerebral disease, and in a severe case of pericarditis. Traube, four years later, alleged that he had witnessed it in connexion with valvular disease at the aortic and mitral orifices, the heart not being fatty; and likewise in cases of cerebral tumors and hæmorrhage, uræmic coma, and tubercular meningitis. He regards the symptom as due to want of arterial

* *Vide* case by Dr. Kidd, quoted in Dr. Ogle's memoir.

† *Vide* Ogle, *loco citat.*

‡ *Clinical Medicine*, 1862, p. 495.

§ *Traube*, 1871.

blood in the respiratory nerve-centres, in consequence of which, respiration failing, and carbonic acid accumulating in the lungs, the pneumogastric nerves, and subsequently the sentient nerves generally, are stimulated to inordinate action, and accelerated respiration is the result. But, carbonic acid being rapidly eliminated under this increased activity of respiration, the peripheral sentient nerves, and then the pneumogastrics, are inadequately stimulated; hence, a gradual subsidence of respiration, and finally apnoea.

Filehne is of opinion that this symptom arises from a lowering of excitability in the respiratory nerve-centre, relatively to that of the vaso-motor centre.*

This form of respiratory derangement is unique. I have never witnessed it save in connexion with an advanced degree of atheromatous change and dilatation of the arch of the aorta, with or without fatty degeneration of the heart; and, judging from my personal experience, such as it has been, I feel bound to regard it as pathognomonic of the conditions of the aorta just mentioned.

Obstruction of the thoracic duct by the pressure of an aneurism has been very rarely witnessed. Laennec has recorded one such example, in which lymphatic engorgement of the lower half of the body was a prominent symptom.† The symptoms arising from erosion of the vertebræ are, on the contrary, of frequent occurrence. Such symptoms are most frequently witnessed in connexion with aneurisms of the descending portion of the arch, the thoracic, and the abdominal aorta. Many examples of absorption of the vertebræ by the pressure of an aneurism of the ascending or transverse portion of the arch, have, however, been recorded; but in these cases pressure upon the intermediate parts, the trachea, the œsophagus, and the great nerve-trunks, necessarily preceded, and gave rise to corresponding symptoms.

Erosion of the vertebræ is accompanied by a fixed "boring" pain. With this are usually associated severe radiating pains of a darting or neuralgic character, but intermittent, and referred

* *Vide résumé in The Irish Hospital Gazette*, November 2nd, 1874.

† *Traité de l'Auscultation Médiate*, tom. ii., p. 714; see also a case by Dr. Hughes Bennett, *Principles and Practice of Medicine*, second edition, p. 567.

to the seat of fixed pain as their centre. The former is due to the action of the tumor upon the proper structures of the vertebral column, and the latter to the spinal compression of nerves, the paroxysms being caused by the temporary expansion of the sac under increased vascular pressure. Spasm, or paralysis of the muscles supplied from the portion of the cord below the seat of pressure, may result from erosion of the vertebra, by irritation or destruction of the corresponding motor nerves. Examples of paraplegia from the irruption of an aneurism of the descending aorta into the spinal canal, have been recorded by Laennec* and Andral,† and another by Dr. Hughes Bennett, from pressure of the sac upon the spinal cord.‡

The sac, pressed against the solid bone, is quickly broken through by the repeated impulse of the blood, the periosteum is next removed by absorption, and, under the remittent pressure of the aneurism, the osseous texture is starved by interruption of its arterial supply, and ultimately disintegrated by a process of molecular necrosis; the *débris* of the bone-substance is mixed with the contents of the sac. The intervertebral fibro-cartilages, owing to their resiliency, are capable of appropriating sufficient pabulum to maintain their vitality, during the brief periods of interrupted pressure corresponding to ventricular diastole.

The *physical signs* of thoracic aneurism may be conveniently classified under the two heads of extrinsic and intrinsic phenomena. The extrinsic signs, which are identical with those of pressure upon the air passages, the lungs, and the laryngeal and bronchial nerves, have been already discussed. The intrinsic signs of aneurism may be now considered under the heads of (*a*) percussion-dulness, (*b*) impulse, (*c*) sounds, and (*d*) thrill.

Dulness on percussion is clinically associated with internal aneurism, only when it has come into contact with the thoracic or abdominal parietes. It is absolute, coextensive with the area of contact, and accompanied by a feeling of resistance communicated to the finger. The extent and the degree of dulness are usually progressive; but, owing to retrocession of the tumor in the course of the disease, percussion resonance may replace dul-

* *Ibid.*, p. 715.

† *Acad. de Méd.*, 1854.

‡ *Opus citat.*, p. 572.

ness. Not only the area, but likewise the location of dulness, may undergo change from the same cause.

The impulse of thoracic aneurism is most frequently single and systolic. In a somewhat less numerical proportion it is double (systolic and diastolic), as first pointed out by Dr. Stokes in 1834.* The systolic or principal impulse is heaving and expansile in character, and coincides with the impulse of the heart; the second is abrupt and non-expansile, diastolic or postdiastolic in rhythm, and communicates to the hand placed over the tumor, a sensation as of sudden arrest to the collapse of the sac towards its termination. From this latter peculiarity it has been designated a "back-stroke;" but as this title implies re-expansion of the sac, it is objectionable. A better designation, because more accurately expressive of the cause and character of the phenomenon, would be an *impulse of arrest*, under which title I shall henceforward refer to it. With the systolic impulse, and coinciding with it in time, is not unfrequently associated fremitus or thrill of a more or less decided character.

The systolic or principal impulse of aneurism is due to the forced expansion of the sac, under the pressure of influx from the main artery at the moment of ventricular systole; hence, it is essentially expansile in character. Thrill is by no means an ordinary accompaniment of aneurismal impulse; when present, it is due to vibration of the sac, either from an eddy in the current of influx, or from a lax state of the sac itself. A spiculated condition of the orifice of the aneurism, or a flake of fibrin partially detached from its walls and floating in the passage, may produce a vortex in the blood-stream, and consequent thrill. The existence of tactile fremitus, therefore, not communicated from the heart, is pathognomonic of vascular tumor of some kind, and where independent evidence of aneurism exists, it may be regarded as in the highest degree confirmatory.

Doctor Bellingham regarded aneurismal *frémissement* as due to regurgitation of blood into the sac during the diastole of the ventricle. That this explanation cannot be admitted, will appear from the fact that the phenomenon in question is *systolic*, not diastolic, in rhythm.

* *Dublin Journal of Medical Science*, vol. v.

The second or diastolic impulse is not of constant occurrence in aneurism, but when present, it is, no less than fremitus, diagnostic of that disease, owing to the peculiar circumstances under which alone it can be produced.

I think Gendrin's explanation of this phenomenon the most rational.* He refers it to want of synchronism between the reaction of the sac and that of the adjacent portions of the artery from which it arises; the artery reacts immediately after ventricular systole, and so causes a second wave to enter the sac, at a moment when, owing to the tardiness of its recoil, from defective elasticity, it is still in process of reaction. Hence, a collision between the volume of influx and that of efflux, and a sudden arrest to the contraction of the sac. If palpable tumor exist, the hand placed over it will perceive a uniform expansion, with an increase of tension coinciding with ventricular systole, which is immediately followed by a "movement of retraction,"† and an impulse of arrest. Pressure upon the artery on the distal side of the aneurism will increase the force of the systolic impulse, but it will abolish the diastolic, by maintaining a state of continuous tension of the sac. Gendrin considers the second impulse to be distinctive of aneurism; in this opinion I entirely concur.

The usual acoustic signs of aneurism of the arch consist in two sounds which coincide with the double impulse when such is present, and closely resemble the sounds of the heart. So close, indeed, is the resemblance, that Laennec regarded the sounds heard in an aneurism close to the heart, as being actually those of the heart itself, propagated to the aneurism. The frequent existence, however, of two sounds in portions of the aorta remote from the heart, renders this doctrine untenable. No less inaccurate is the unqualified statement of Hope, that the first sound of an intra-thoracic aneurism "is a murmur."‡ The existence of murmur in thoracic aneurism is the exception.

The sounds of aneurism are usually louder than those of the heart in the same person; a circumstance attributed by Bertin to the echo of a large cavity, as in pectoriloquy. This is espe-

* *Revue Médicale*, 1844, tom. iii., p. 508.

† Gendrin, *loco citat.*

‡ *Opus citat.*, p. 442.

cially true of the second sound, which, as Dr. J. W. Begbie avers, is accentuated in thoracic aneurism and in simple dilatation of the aorta. In aneurism, too, the second sound frequently possesses a "booming" character.* Dr. George W. Balfour holds a similar opinion in regard to the significance of an accentuated second sound in the aorta.† Both these writers believe that the phenomenon is due to the recoil of an increased volume of blood upon the valves. This subject has been discussed in a previous chapter.‡ I will here only repeat, that in my opinion, an intensified aortic second sound indicates atheroma and dilatation of the aorta with hypertrophy of the left ventricle. It may, therefore, exist in connexion with aneurism of the arch in which these conditions are supplied, quite irrespectively of its locality. The second sound in the aorta may be sharp and clear, without being intensified; it may, likewise, possess a "booming" quality. The former of these characteristics may be exhibited in aneurism; the latter is peculiar to it.

The aneurismal sounds have been attributed to various causes. Gendrin believed that they are due to the shock of impulse exclusively; he accordingly designated them as the first and second "*bruit de choc*," respectively. To Gendrin's theory of the second impulse and sound in aneurism Bellingham objects: (*a*) if it were true, a second impulse and a second sound should exist in the aneurism of minor arteries, but such is not the case; and (*b*) the adjacent portions of the vessel are assumed to be sound, whereas they are usually unsound.§ To these objections it may be answered, that the elasticity of the secondary arteries is much less than that of the aorta, and that the walls of the aorta in the vicinity of the aneurism are shown to be functionally competent by their not being aneurismal.

Bellingham attributed both the sounds of aneurism to the friction of the blood against the orifice of the sac; the first, to

* *Edinburgh Medical Journal*, vol. viii., 1862-3. Dr. Stokes had, eight years previously, mentioned a "ringing" second sound as characteristic of thoracic aneurism (*Diseases of the Heart and Aorta*, 1854, p. 539). He was, therefore, the first to call attention to this sign.

† *Edinburgh Medical Journal*, January, 1874.

‡ *Vide* Chapter ii., p. 138.

§ *Dublin Medical Press*, June 7th, 1848.

that of influx during ventricular systole, and the second, to that of regurgitation into the sac from the vessels beyond.* To the last mentioned cause, namely, passive regurgitation of blood into the sac during ventricular diastole, under the influence of gravity, he likewise attributes the second impulse in thoracic aneurism. Against this latter doctrine several objections may be urged, which may be briefly stated as follows. It would necessarily imply the coexistence of the visible throbbing of "unfilled arteries" in the neck, and regurgitant murmur in the sac, as remarked by Dr. Stokes, phenomena rarely witnessed in connexion with thoracic aneurism. As observed by Dr. Lyons, lowering the head to the level of the body, the patient being recumbent, will not modify, much less suppress, the second impulse and sound, where both exist, in thoracic aneurism, nor, in femoral aneurism with a single impulse, will the elevation of the leg above the level of the trunk develop a second impulse, as proved experimentally by the same observer. Finally, it implies the absence of vascular reaction in sound arteries, and ignores the reaction of the arch of the aorta. A force by which the closure of the valves is effected and a second sound is produced, must be competent to prevent the gravitation of a column of blood from the arteries of the neck; hence, the second impulse and sound of aneurism could not, on Bellingham's theory, coexist with a second cardiac sound. But their concurrence is certainly the rule.†

Doctor Lyons, in a very able article, has propounded a theory of the second impulse in aneurism, the existence of which he would justly regard as proof that the aneurism was saccular and belonged to the "false" variety.‡ He agrees with Gendrin in attributing the second impulse of the sac in thoracic aneurism, to the influx of a second volume of blood under the reaction of the adjacent portions of the aorta. The absence of a second impulse in abdominal aneurism, and in aneurism of the extremities, he accounts for by assuming a fusion at a certain point of the aorta, probably at the commencement of the abdominal por-

* *Ibid.*

† This subject has been already referred to at p. 844, in connexion with the signs of dilatation of the aorta.

‡ *Dublin Quarterly Journal of Medical Science*, vol. ix., 1850, p. 319.

tion of the vessel, of the two propelling forces by which the blood is driven forward, namely, those of ventricular systole and arterial reaction; "that of the ventricular systole, being, as it were, overtaken by that of the arterial." To the want of concurrence of these two forces in saccular aneurism, the second impulse is, in his opinion, due; and to their fusion in aneurisms below the diaphragm, the absence of a second impulse is to be attributed. Against this theory, which would afford the most rational explanation that has been yet offered of the absence of a second impulse in abdominal aneurism, the objection may be urged that, carried to its legitimate conclusion, it would imply that the pulsation of abdominal aneurism, and the ordinary pulsation of distant arteries, could never precede the second sound of the heart, inasmuch as one of the assumed factors of such pulsation, the wave of aortic reaction, must necessarily be posterior in time to the second sound. It is, however, well known that both the phenomena referred to usually take precedence of the second sound.

I believe shock to be the chief factor in both the aneurismal sounds; whilst the density of the sac and surrounding structures accounts for their comparative intensity. I cannot pretend to explain the "booming" quality which occasionally distinguishes the second sound in aneurism, except on the questionable hypothesis of a momentary extrication of gas within the sac during ventricular diastole. On the cessation of systole, the sac becomes relaxed by reflux of the blood within it; into the virtual vacuum so produced, it is not inconceivable that gas may, for the moment, be evolved, to be again taken up by the blood under the increased pressure of the succeeding systole of the ventricle. Bernard has found that, during digestion, arterial blood contains 5 to 6 per cent. of free carbonic acid, which is readily evolved *in vacuo*. The proportion gradually decreases during the interval between meals, and after twenty-four hours' fasting, not a trace of carbonic acid is to be found in the arterial blood.* It would be interesting to determine whether the "booming" sound of aneurism is in any degree influenced by digestion.

* Quoted by Flint (*Physiology of Man*, p. 464, foot note), from unpublished lectures delivered in 1861.

Gendrin holds that true aneurism is distinguished by a single systolic sound ; whereas, in false aneurism of the large arteries, the sound is double. This doctrine is, however, inadmissible. True aneurism is rarely met with except in the ascending aorta, and here it yields two sounds, which are those of the heart, reinforced by the density of the arterial walls; whilst false aneurism not unfrequently yields a single and systolic sound only.

Murmur is of much less frequent occurrence in thoracic aneurism than impulse or sound. It is usually single, systolic, and blowing, but it may be "booming" in quality.* The murmur of thoracic aneurism is, however, occasionally double, and still more rarely, a diastolic murmur only exists. Dr. Bellingham has published the details of 12 cases of thoracic aneurism, in all of which a double sound, normal or abnormal, was present; in most of these cases a double impulse likewise existed; in 2 instances a murmur accompanied or replaced the first sound: in one, murmur replaced both sounds, and in one, a murmur replaced the second sound. In 8 of these cases a double sound, closely resembling the sounds of the heart, existed.† With reference to this point, the following evidence is furnished by Table XVI. Impulse was observed in 29 cases; it was single and systolic in 22, and double in 7; it did not exist in 32 cases; and was not referred to in 7. The presence or absence of aneurismal murmur was noted in 61 cases; of these, it existed in 20; in 16 of this number it was single and systolic, and in one it was single and diastolic; it was double in 3 instances, and was absent in 41 cases.

The presence or absence of aneurismal sound, as distinct from murmur, was noted in 61 cases; it was present in 11 instances, in 2 of which it was single and systolic, and in 9 it was double. Sound was not present in 50 cases. A double impulse and a systolic sound coexisted with a diastolic murmur in a single instance. The existence of a diastolic murmur in aneurism is very rare; I have not met with an example of it. In one instance only I have noted a murmur of diastolic rhythm in *connexion* with aneurism of the ascending portion of the arch; but

* *Vide* Case 69, p. 759.

† *Dublin Medical Press*, April 19th, May 24th, and June 7th, 1848; see also Dr. Greene's cases in *The Dublin Journal of Medical Science*, vols. vii. and x.

in that case it was obviously due to valvular inadequacy from dilatation of the aorta.* In the solitary instance (No. 45), in Table XVI., in which diastolic murmur existed, the aneurism was likewise connected with the ascending aorta. In another case (No. 60) a diastolic murmur was occasionally heard in connexion with an aneurism of the thoracic aorta. I must, therefore, regard diastolic murmur as an exceptional phenomenon in aneurism. It is, I believe, in most cases due to aortic regurgitation; but that it is occasionally intrinsic to aneurism must be admitted.

The murmurs of aneurism arise from the same causes as those of the heart, namely, friction and vibration. The doctrine of unequal pressure and velocity of current arising from abrupt constriction of a conduit, as first propounded by Corrigan, subsequently developed by the experiments of Chauveau, and by those of Dr. Leared,† may be studied with advantage in reference to this subject, which has been already discussed at page 181 and page 258.

As principal or contributory causes of aneurismal murmur, I may briefly advert to: (a) the figure, the relative diameter, and the actual state of the orifice and the walls of the sac; (b) the contents of the sac; and (c) the force of ventricular contraction. A relatively narrow orifice with a rough margin, a globular and flaccid sac springing from the vessel at an angle and containing fluid blood, and vigorous contraction of the left ventricle, constitute the most favourable conditions for the development of murmur.

Of these, the last mentioned is the principal factor of murmur; hence, at the approach of death, murmur, whether cardiac or aneurismal, becomes indistinct or inaudible, from failure of the heart. The absence of one or more of the other conditions mentioned may modify or exclude murmur, even though the heart act with energy.

Thoracic aneurism may be now considered in its clinical aspect, with a view to differential diagnosis. It may present, in a

* Case 141.

† *Medical Times and Gazette*, October 31st, 1868. Dr. Leared has expounded his theory in an earlier publication, a reference to which I have not been able to find.

more or less modified form, a combination of the foregoing symptoms and signs, or afford evidence of so indefinite a character that it may be practically regarded as latent.

The existence in the chest of a centre of pulsation, distinct from that of the heart, would suggest aneurism, cancerous tumor, or pulsating empyema. The presence, in such centre, of a double sound, the second being louder than that of the heart, would, *pro tanto*, be in favour of aneurism, and tend to exclude cancer and empyema. A double impulse, the second being an impulse of arrest, would possess a similar but stronger significance. Single and systolic murmur would indicate aneurism and carcinoma in about equal proportion, and would exclude empyema. A double murmur, if associated with abnormal impulse in the situation of the ascending portion of the arch, would suggest aneurism rather than cancer or empyema.

The symptoms of excentric pressure, considered apart from other evidence, have no distinctive significance save in regard to their inconstancy or variation. If, in the progress of the case, symptoms of this character cease to be exhibited, or vary in degree, the presumption would be in favour of aneurism. The occurrence of paroxysmal dyspnœa, laryngeal or tracheal, associated with excitement of the circulation from physical or emotional causes, would likewise point to aneurism. But even in this respect, an abscess located between the trachea and œsophagus, may simulate aneurism. The following example came under my own notice.

A man, aged fifty, was admitted into the Mater Misericordiae Hospital on the 13th of April, 1864. He was in a state of imminent asphyxia, with cold extremities, lividity of surface, and a loud laryngeal stridor.* The pulse was rapid, feeble, and intermittent, and deglutition, even of liquids, was attended with the greatest difficulty. The chest was universally resonant, and no evidence of pulmonary or vascular disease could be detected. Tracheotomy was performed on the evening of the 13th, and was followed by complete relief from all the urgent symptoms. On the 18th he had another paroxysm of dyspnœa which nearly proved fatal. On being raised in bed he coughed up about a

* *Proceedings of the Pathological Society of Dublin*, vol. ii., part ii., p. 143.

pint of purulent matter, and got immediate relief. Loud bronchial râles were audible all over the chest. He could now swallow solid food. He had a third paroxysm on the 20th, and he died worn out on the 22nd. The viscera were healthy; a large abscess, which had been recently filled with pus, but was now empty, was found between the trachea and œsophagus. It extended from the larynx downwards into the posterior mediastinum, where it ended in a *cul de sac*. The arytenoid cartilages were necrosed; they were all but detached by ulceration from the cricoid, and had fallen forward upon the glottis. A large, ulcerated opening extended from the œsophagus into the abscess, whilst another and much smaller aperture connected the abscess with the trachea. Through the former opening, which had evidently been formed by a perforating ulcer of the œsophagus, particles of food had escaped and given rise to the abscess, whilst the paroxysms were the result of spasm from pressure upon the trachea, and latterly, of dislocation of the arytenoid cartilages.

An example of laryngeal distress and dysphonia, with immobility of the vocal chord, from paralysis of the right recurrent laryngeal nerve produced by the pressure of a malignant tumor of the œsophagus, has been published by Dr. Morell MacKenzie.*

If, on the other hand, the signs of persistent excentric pressure within the chest coincide with cancerous tumor developed externally, the evidence, so far, would be in favour of intra-thoracic cancer.

Pulsating cancer is further distinguishable by the fact that the force and diffusion of impulse are not in proportion to the degree and extent of dulness; nor does the centre of pulsation correspond to that of dulness; and, further, the superficial thoracic veins on the side affected by intra-thoracic cancer are usually enlarged and varicose, whilst those related to an aneurism are never varicose, although they may be distended.† Pulsating emphysema, of which three remarkable examples have been published by Dr. Robert L. MacDonnell,‡ and one by Dr. Stokes,§

* *Lancet*, May 30th, 1874.

† Vide Stokes in *Dublin Journal of Medical Science*, vol. xxi., p. 227. Also a case by Dr. O'Ferrall, *Proceedings of the Pathological Society*, April 23rd, 1842.

‡ *Ibid.*, vol. xxv., p. 1.

§ *Diseases of the Heart and Aorta*.

may be distinguished by its occupying the normal position of the heart, which is displaced to the right side, by its being preceded by the symptoms and signs of acute inflammation on the left side of the chest, and by the presence of a single and systolic impulse communicated from the heart, and unattended with murmur, sound, or thrill. The foregoing evidence, positive and negative, will suffice to distinguish pulsating empyema from aneurism, even in the absence of metallic phenomena in the left side of the chest, which would be in themselves conclusive.

Laryngitis may be mistaken for thoracic aneurism, owing to the attendant symptoms of hoarseness or aphonia, dyspnoea, and stridor. The distinction would rest mainly upon the positive result of laryngoscopic inspection, and the absence of other symptoms, and of all the intrinsic signs of aneurism. The opposite and more serious error, that of mistaking aneurism for laryngitis, may be likewise committed. Dr. Gairdner mentions a case in which this mistake was actually made by an expert in laryngeal disease; and quite recently a similar case was published in which tracheotomy was actually performed with a fatal result. In this case the aneurism was latent, and the operation was demanded by the urgency of the symptoms.

Neuralgia or rheumatism engaging the muscles of the chest, owing to the attendant symptom of radiating or fixed pain, may be likewise confounded with aneurism; so also may tuberculosis and cirrhosis of the lungs, from the occurrence of hæmoptysis. But if a suspicion of aneurism be once aroused, the distinction from these diseases cannot be difficult. An error of diagnosis can arise only from the idea of aneurism *not* having been presented to the mind of the physician.

Doctor George W. Balfour has recently pointed out another possible source of error in the diagnosis of thoracic aneurism.* A systolic murmur, with visible pulsation localized in the inner portion of the left second intercostal space, may arise from mitral reflux of anæmic or organic origin, propagated into the left auricular appendix, as first mentioned by Naunyn; or a presystolic murmur from mitral narrowing, similarly propagated,

**Medical Times and Gazette*, December 12th, 1874.

and associated with impulse, may be perceived in the same situation. An impulse and murmur of systolic rhythm, and located in the pulmonary artery, may be likewise presented in the second interspace, but closer to the sternum, as first pointed out by Quincke. It was attributed by him to flattening of the pulmonary artery against the anterior wall of the chest during ventricular systole, the left lung having been permanently retracted from antecedent disease. Dr. Balfour details four cases in which these phenomena were exhibited. He observes, however, that the distinction from aneurism was readily made in all these cases by the absence of signs of excentric pressure, and by the more feeble impulse at the second centre of pulsation, as compared with that of the heart.

In an earlier publication on the same subject, Dr. Balfour had suggested a very ingenious means, by which, having reference to the relative rhythm of the cardiac and the abnormal pulsation, he could readily determine whether the latter was auricular or arterial. It consists of two pellets of bees' wax, with a bristle, supporting a small paper flag, fixed in each. One of these pellets is attached over the apex of the heart, and the other at the seat of abnormal pulsation. If the superior pulsation precede the inferior, as indicated by the movement of the flags, it would be regarded as auricular; if it accompany or succeed the inferior pulsation, it would indicate aneurism or pulsation in the pulmonary artery. The distinction between the two latter should be made by reference to the signs of excentric pressure, and relative force of pulsation, as already mentioned.*

On Quincke's theory, murmur should always accompany visible pulsation of the pulmonary artery; but such is not the case. I would further remark, that the murmur of mitral stenosis is not usually audible in the left second interspace, although the attendant pulsation of the left auricular appendix is frequently visible there. But the presystolic rhythm of both these phenomena would, in any case, suffice to exclude aneurism.

The general symptoms and signs most distinctive of thoracic aneurism are: a pulsating tumor yielding a single impulse and a double sound, distinct from those of the heart, the second

* *Edinburgh Medical Journal*, January, 1874.

impulse being a back-stroke, or an impulse of arrest, and the second sound louder than that of the heart; paroxysmal or remittent pains of a radiating or shifting character; and the evidence of excentric pressure upon adjacent parts. None of these are, however, of constant occurrence, even in aneurisms having the same origin and relationship. Hence, the diagnosis must rest upon a rational basis, and be deduced from a careful consideration of *all* the features presented in each case.

The limits of variation are pretty well defined in regard to (*a*) true, (*b*) false, and (*c*) varicose aneurism, respectively; they are also, but less definitely, fixed in relation to the portion of the aorta whence the aneurism has arisen. It will be therefore necessary to review briefly the semeiology of thoracic aneurism under each of the foregoing heads.

True aneurism is rarely met with except in the ascending portion of the arch; it is tubular or fusiform, and cannot attain large dimensions without undergoing conversion into the false variety, by rupture of its internal tunics. Owing to this circumstance, and the usual absence of clot or deposited fibrin, true aneurism is characterized by few and indefinite signs. Diastolic impulse, murmur, or thrill, are seldom exhibited, because the necessary conditions for these phenomena, lateral deviation of current, and roughness of surface, are not present,* and single and systolic bruit is not a sign of true aneurism, as stated by Bertin. Hence, the diagnosis of true aneurism must be in the highest degree doubtful. Indeed, the only positive sign which it may exhibit is an accentuated second sound, but this is equally characteristic of simple dilatation with atheromatous rigidity of the aorta.

The symptoms exhibited by a false aneurism within the chest, irrespectively of intrinsic pain, will depend upon the organs or parts implicated in its growth; these have been already considered in detail. I shall only add the following clinical remarks suggested by the cases herewith given.

* Cruveiller holds that clotting cannot take place in an artery without previous erosion or inflammation of its internal surface; and F. Niemeyer (*Medical Times and Gazette*, January 22nd, 1870), declares that roughness of the *intima*, constituting *endarteritis deformans*, is an essential condition of fibrinous deposit in an artery.

Case 136, shows that pressure upon the left bronchus, amounting nearly to occlusion, need not necessarily cause dysphonia, laryngeal cough, stridor, or paroxysmal dyspnoea; that vocal vibration may exist in a lung from which air has been all but excluded by pressure upon the primary bronchus of that side, but that it is abolished by solidification of a lung so circumstanced; and finally, it shows that an aneurism may open simultaneously into the oesophagus and one of the bronchi.

Case 138, shows that saccular aneurism, with a large and smooth-edged orifice, an empty cavity, and flaccid walls, may present no intrinsic physical signs, and if small, deep-seated, and remote from the walls of the chest, may afford no evidence of its existence save that arising from excentric pressure; and that the most aggravated dyspnoea and stridor, from spasm of the larynx, may be due to reflex irritation of the recurrent laryngeal nerves. It also proves that feeble respiration in one lung, accompanied by laryngeal spasm, may be caused by pressure upon the trachea, and that, in such cases, laryngotomy may be followed by complete relief from urgent symptoms.

With regard to relative frequency of occurrence, in combination and singly, the various tactile and acoustic phenomena of thoracic aneurism, as deduced from the 17 cases (comprising 18 aneurisms*) which I have personally noted, might be set down in the following order:

(a) Double impulse and double sound, without murmur	...	3 cases.
(b) Systolic impulse and double sound, without murmur	...	4 „
(c) Systolic impulse and systolic murmur, without sound	...	3 „
(d) Double impulse and double sound, with systolic murmur	...	1 „
(e) Systolic impulse and double murmur, without sound	...	1 „
(f) Double murmur, without impulse or sound	...	1 „
(g) No impulse, sound, or murmur	...	5 „
		— 18

It will thus be seen that:

Double sound existed in	8 cases.
Single and systolic impulse in	8 „
Double impulse in	4 „
Single and systolic murmur in	4 „
Double murmur in	2 „

and that, in 5 out of the 17 cases, the aneurism was latent, *quoad* tactile and acoustic signs.

* In one case (No. 150) there were two aneurisms.

Varicose or *communicating* aneurism is defined by Stokes as that variety of the disease in which "blood passes from one portion of the vascular system to another." Aneurism within the chest may establish communication with (a) the chambers of the heart, (b) the pulmonary artery, or its branches, and (c) the descending cava, or the innominate veins.

According to Peacock,* the first recorded example of varicose aneurism was one opening into the left auricle, and published by Beauchene in 1811. Wells, in 1812, published a case in which an aneurism of the aorta formed a communication with the pulmonary artery. In 1832 an example of varicose aneurism implicating the superior vena cava, was recorded in *The Lancet*.

Cases were subsequently recorded by Curling (1838), Munro, (1839), Reid (4 cases), and Thurnam (10 cases, including the preceding, 1840). Peacock gives reference to 33 cases, not including 6 referred to by Thurnam, as contained in various museums, and 4 met with by Rokitansky.

In 18 of this number, the aneurism arose from one or more of the sinuses of Valsalva or from the aorta immediately above the sinuses; in 12, from the ascending portion of the arch, above the points mentioned; in 2, the aneurism engaged the entire arch except the termination of its descending portion; and in one case there were two aneurisms, one in the ascending, and another in the descending portion of the arch, the latter opening into the left branch of the pulmonary artery.

Only two examples of varicose aneurism have come under my notice. In one of these, the aneurism opened into the right ventricle (Case 69, p. 759), and in the other (Case 141), into the superior cava. Two additional examples have been recently recorded in *The Proceedings of the Pathological Society of Dublin*; one by Dr. Gordon, in which an aneurism of the left anterior sinus of the aorta opened into the left ventricle of the heart,† and another, by Dr. Bennett, in which an aneurism of the right sinus communicated also with the left ventricle.‡

Doctor McDowel exhibited before the Pathological Society, in January, 1849, an example of varicose aneurism springing from the aorta, half an inch above the valves, and opening into

* *Transactions of the Pathological Society of London*, vol. xix., p. 111.

† Vol. ii., part i., new series.

‡ Vol. iii., part ii.

the right auricle. The tumor pointed externally in the right subclavicular region, and yielded a systolic impulse, accompanied by fremitus, and a harsh systolic murmur audible only between the middle line and the right nipple.

Hope, in 1839, published a remarkable case of aneurism of the aorta immediately above the valves, communicating with the right ventricle by two apertures. The symptoms and signs, which followed a strain in lifting a weight, and a "creak in the heart," accompanied by faintness, were lividity, cedema, and a jerking pulse, purring tremor, double murmur, and a continuous rumble to the left of the sternum, from the second to the fourth rib.*

In the case which came under my own notice, through the favour of Dr. Stokes, an aneurism of the size of a tennis ball, arising from one of the aortic sinuses, communicated with the right ventricle by an aperture of the size and figure of a shirt button-hole. The symptoms were, general venous congestion with cedema; and the physical signs, *frémissement*, with a loud and booming double murmur at midsternum, audible to the patient, and compared by him to "a woman churning;" all which signs were subsequently presented with greatest intensity at the apex of the heart, but diffused over the entire left front of the chest. The diagnosis of aneurism opening into one of the ventricles was presumptively made.†

Thurnam has included in his list of cases of varicose aneurism three examples of aneurism of the aorta opening into the heart. In two of these cases, observed by Curling and Reid respectively, a communication was established with the right auricle; and in the third, supplied by himself, with the right ventricle by two apertures. In this case, a loud superficial and continuous sawing murmur, accompanied with purring tremor, was audible in the left second intercostal space, an inch and a-half from the sternum, but loudest over a space not larger than a shilling. The heart had been displaced to the left by effusion into the right pleural cavity.‡

Aneurisms projecting into the chambers of the heart, but not communicating with them, must be distinguished from those

* *Diseases of the Heart and Great Vessels*, third edition, p. 466.

† *Vide Case 69*, p. 759.

‡ *Medico-Chirurgical Transactions*, vol. xxiii, p. 323.

which have established such a communication. The symptoms and signs must obviously differ in the two classes of cases; hence the necessity for a clinical distinction between them.

Professor Smith's case was one of the earliest recorded examples of this kind. A man, aged thirty, was admitted into hospital suffering from urgent dyspnoea, which was accompanied by lividity, œdema, and suppression of pulse. There was great extension of precordial dulness, with feeble action of the heart. He died on the following day. An aneurism, with an orifice as large as a shilling, arose from the aorta one inch above the valves, and extended downwards behind the pulmonary artery, which lay across it, and gave the appearance of two tumors, each as large as a hen-egg. One of these projected into the base of the right ventricle near the pulmonary orifice, and the other into the superior and anterior portion of the left ventricle. The aneurism did not communicate with either ventricle.*

An example of aneurism of one of the sinuses of Valsalva, projecting into the right auricle, was presented to the Pathological Society of Dublin by Dr. Jennings, in December, 1866.

Doctor Law exhibited before the same Society in February, 1870, an example of aneurism springing from the posterior sinus, advancing downwards and to the right, displacing the liver and causing slight jaundice. It projected into the right auricle of the heart, occluding the orifice of the inferior cava, and partially closing that of the superior. The most prominent symptoms were general venous congestion and dropsy.

Doctor Hanna has published a case in which an aneurism, as large as an orange, arose from one of the sinuses of Valsalva by an orifice two lines in diameter, and was imbedded in the wall of the left ventricle. The muscular substance had been entirely absorbed around the sac, which was formed by the thickened membranes of the heart, and contained laminated fibrin. The patient was a man, aged thirty-one, who had sustained a severe fall from a horse, and shortly afterwards complained of severe pain in his chest, palpitation, and dyspnoea; he also spat blood. The symptoms were orthopnoea, and feeble but regular pulse. In the left second interspace there was a double impulse accom-

* *Dublin Journal of Medical and Chemical Science*, vol. ix. See also a case by Professor Harrison, *ibid.* vol. xv.

panied by thrill, and a whirring noise. These signs existed throughout the precordium, but were most distinct in the second intercostal space.*

Varicose aneurism between the aorta and the pulmonary artery is characterized by symptoms and signs nearly identical with that variety of the disease examined in the preceding paragraphs. It may therefore be discussed here. Dr. Thurnam has collected from various sources eleven examples of this variety of aneurism, and of five of these he has given a full history.†

A case is given in detail by Professor Smith, which may be briefly referred to. A man, aged twenty-two, had been subject to vertigo with temporary loss of vision, dyspnœa, faintness, and chills; then followed general venous congestion, cough, orthopnœa, anasarca, and intermitting pulse. He was troubled by great anxiety and disturbing dreams. There was extension of precordial dulness, with tumultuous action of the heart, *frémissement* which was perceptible through the bed-clothes, and systolic murmur. The man felt "as if there was a living bird in his chest." He subsequently had epileptic seizures, in one of which he died. The lungs were congested, and the aorta communicated with the pulmonary artery, near its origin, by an opening with thick and rounded edges.‡

Doctor Peacock has published a case in which an aneurism of the ascending aorta opened into the pulmonary artery by an orifice two lines by four in diameter; it projected also into the right ventricle, destroying one of the pulmonic valves. The heart was enlarged, and the aortic valves were disorganized. The symptoms were dyspnœa, general lividity, and cedema of the feet. There was extension of precordial dulness, with systolic murmur all over the precordium, and postdiastolic murmur at the right base. The second sound was very sharp in the left second interspace near the sternum. This latter phenomenon he attributed to the tension of the sac during diastole.§ In a case by Dr. W. Wade, the diagnosis was made from the existence of a basic diastolic murmur, not due to inadequacy of the aortic valves.||

* *Ibid.*, March 1st, 1835. † *Loco citat.*

‡ *Dublin Journal of Medical Science*, vol. xviii. See also the case given by Hope, *opus citat.*, p. 469.

§ *Transactions of Pathological Society of London*, vol. xix., p. 111.

|| *Medico-Chirurgical Transactions*, vol. xlv.

An aneurism may compress the pulmonary artery without communicating with it. The two classes of cases are characterized by special clinical features, and should not be confounded.

An example of compression of the pulmonary artery by an aneurism of the ascending aorta has been published by Sir D. Corrigan. In addition to the ordinary signs of aneurism, there was general venous congestion, with dropsy and delirium.*

Of aneurism, communicating with either vena cava, several examples are on record. One of the earliest was that published by Mr. Syme, in which an aneurism of the size of an orange, arising from the abdominal aorta above its bifurcation, opened into the inferior cava by an aperture as large as a sixpenny piece. There was œdema of the lower half of the body; also a loud whirring noise audible to the patient and his friends.†

Doctor Thurnam has published‡ a case of a large aneurism of the ascending aorta, opening into the superior cava by an orifice a quarter of an inch in diameter. There was cyanosis, with œdema of the upper half of the body, and a loud buzzing noise over the vena cava. This case was observed by Dr. Thurnam in 1833. The next, and the most important case of this kind, because a positive diagnosis of the lesion was made from the phenomenon exhibited, has been recorded by Dr. Mayne.§ A large aneurism, engaging the entire arch of the aorta, had compressed and occluded both innominate veins and the superior cava, with the latter of which vessels it had formed a communication by an opening of the size and figure of the button-hole of a shirt, crossed by a delicate band. The patient, a woman aged fifty, whilst washing in a stooping posture, was suddenly seized with symptoms of strangulation, accompanied by dizziness. Her face became livid; her breathing embarrassed. When the patient was seen by Dr. Mayne, the upper half of the body was puffed and deeply cyanosed, the eyes protruded, the veins of the neck

* *Lancet*, February 7th, 1829.† See also a similar case by Mr. Carmichael, *Proceedings of Pathological Society of Dublin*, December 5th, 1840.

† *Edinburgh Medical and Surgical Journal*, vol. xxxvi., 1831, quoted by Thurnam (*loci citat.*), who also refers to two other similar cases, one by Mr. Robinson, of Camberwell, and another by Dr. John Reid; in neither of which, however, was a stethoscopic examination made.

‡ *Loco citat.*

§ *Dublin Quarterly Journal of Medical Science*, November, 1853.

and upper extremities were greatly distended, whilst those of the lower half of the trunk and the lower extremities, which were quite free from cedema, and of their natural colour, were in no degree congested. The pulse was jerking. The physical signs were a heaving systolic impulse accompanied by fremitus, and a whirring systolic murmur, immediately to the right of the sternum at the level of the second rib. This murmur was diffused all over the front of the chest; but its point of greatest intensity corresponded to the junction of the second costal cartilage with the sternum, whilst the fremitus was transmitted into the great veins of the neck. The heart was perfectly healthy.

In the case (141) which came under my own observation, there was great venous turgescence, with deep cyanosis and depressed temperature of the upper half of the body, systolic impulse, thrill, and murmur in the seat of the aneurism; the latter transmitted into the carotids. A diastolic murmur likewise existed at mid-sternum, but it was not audible at the seat of impulse and systolic murmur. There was no visible pulsation or thrill of the cervical arteries or veins.

The symptoms and physical signs exhibited in the foregoing cases resolve themselves into two groups; namely, 1st, those which are characteristic of an aneurism communicating with (a) one or more of the chambers of the heart, (b) the pulmonary artery or one of its branches, (c) one of the venæ cavæ; and, 2nd, those which arise from the intrusion, without rupture, of an aneurism into the chambers of the heart, or into one of the vessels just named. The cases in each of these groups are marked by special features sufficiently distinctive of their nature.

The irruption of the contents of an aneurism into the heart, or one of the great vessels named, is usually associated with a definite strain or shock. It is attended with a feeling of suffocation and faintness, and is immediately followed by great dyspnoea, feeble and fluttering pulse, venous congestion more or less general according to the point of abnormal influx, with coextensive anasarca and depression of temperature. The physical signs developed are, thrill associated with impulse, and a harsh, prolonged, systolic murmur of a buzzing character at the seat of aneurism, and transmitted to a variable distance in the line of

the abnormal current. A second murmur, of diastolic rhythm, frequently coexists with the former. When this is the case, the two murmurs, owing to the presence of an intervening rumble, not unfrequently seem to be continuous. The existence of arterial throbbing in the neck, and a jerking pulse at the wrists, depends upon reflux into a large aneurismal reservoir during diastole; it therefore may, or may not, according to the size and capacity of the sac, characterize an arterio-cardiac or an arterio-venous aneurism. The phenomena, then, which distinguish varicose aneurism, viewed in the abstract, are three in number; namely, extreme venous congestion suddenly developed; thrill; and a buzzing murmur of systolic rhythm transmitted in the line of abnormal circulation.

Aneurism communicating with the heart is not characterized by any symptoms or signs by which it may be distinguished from other forms of varicose aneurism within the chest. A sudden transfer of the seat of murmur and thrill, from the base to the apex of the heart, as occurred in Case 69, might be regarded as proof of the irruption of an aneurism into either ventricle.

Aneurism opening into the pulmonary artery is especially characterized by sudden and most urgent dyspnoea, unaccompanied by spasm or stridor, with bloody expectoration. Where aneurism of the ascending portion of the arch of the aorta had been previously verified, the sudden occurrence of these symptoms might be regarded as pathognomonic of a communication between it and the pulmonary artery. Death, in such a case, may occur very rapidly by asphyxia.

The symptoms which distinguish arterio-venous aneurism are those which result from the sudden introduction of retrograde arterial pressure into a definite portion of the venous system. The veins, distal and tributary to that with which the aneurism has established a communication, are engorged, and the corresponding portions of the body are cyanosed and depressed in temperature, whilst all other portions of it retain their normal colour and heat. Hence, a varicose aneurism of the abdominal aorta and the inferior cava is distinguished by venous congestion, cyanosis, and cedema of the lower portion of the

trunk and the inferior extremities, as occurred in Mr. Syme's case already referred to; and direct intercourse between the arch of the aorta and the superior cava, by a similar condition of the upper half of the trunk, the upper extremities, and the head and neck, as illustrated in the cases published by Thurnam, Mayne, and myself.*

Aneurisms communicating with the heart, the pulmonary artery, or the superior cava, have, without an exception known to me, taken origin from the ascending portion of the arch of the aorta.

The simple projection of an aneurism of the aorta into one or more of the chambers of the heart, is attended only with symptoms of obstructed circulation proportionate to its interference with the passage of the blood, and by acoustic signs having reference to its effect upon the orifices and valves.

The modification of the symptoms and signs of thoracic aneurism, dependent upon its position in the chest, may be now considered. The posterior wall of the chest is rarely affected by an aneurism of the first portions of the arch of the aorta. Not unfrequently, however, a large aneurism of the upper portion of the ascending aorta approximates the posterior thoracic wall, and is perceptible in the right scapular region by modified impulse, dulness, and sound. It is still more frequently discoverable in this situation by the respiratory and vocal phenomena arising from compression of the lung; namely, bronchial or tracheal respiration, bronchophony, and vocal fremitus.†

Aneurisms of the descending portion of the arch, and those of the descending thoracic aorta, directly implicate the posterior wall of the chest on the left side in a very large percentage of cases. Indeed, aneurism of the last mentioned portion of the vessel rarely fails to penetrate the posterior wall on that side, and to present a visible tumor in the scapular or infra-scapular region. Erosion of the vertebræ and the ribs are amongst the ordinary consequences of aneurism of these portions of the aorta. Hence, the two kinds of pain, paroxysmal or intermittent, and fixed or boring, already mentioned. The boring pain of vertebral erosion is always associated with local tenderness to per-

* See pp. 1126 and 1127.

† *Vide* Case 142.

cussion over the corresponding spinous processes. It may not, however, have been at any time present, even where extensive destruction of the vertebræ has been effected;* but most certainly its absence is in the highest degree exceptional under such circumstances.

The existence of a double sound over a limited portion of the vertebral column, especially if associated with pain and tenderness in the same situation, is, in the highest degree, suggestive of aneurism; because, as well observed by Laennec and Bertin, the two sounds of the heart are rarely audible in the back. A perceptible impulse on either side of the dorsal spine is likewise suggestive of aneurism; and, owing to the normal position of the thoracic aorta to the left of the vertebral column, such impulse would possess more significance if felt upon the right side, as remarked by Dr. Greene. Darting or shooting pain in the line of the ribs, is a common result of pressure upon the intercostal nerves.

Displacement of the lungs, the heart, or the liver, may result from the growth of an aneurism within the chest. The left lung may be displaced upwards, and condensed, and local pleuritis with friction sound, produced by the pressure. The heart may be likewise dislocated upwards,† forwards,‡ or backwards. Displacement of the heart forwards is characterized by the "double jogging" impulse of Hope; and, in the case mentioned by Bellingham just referred to, it was associated with diastolic murmur which ceased after the retrocession of the heart. In the case recorded by Dr. Mayne, the cardiac impulse was violent, and its area was extended. In a case published by Dr. Douglas Powell, a small aneurism arising from the aorta, one inch above the valves, had descended in front of the heart, displacing the apex backwards.§

Amongst the symptoms especially suggestive of aneurism is a harsh and loud cough of metallic resonance. Even in the absence of stridor, dysphonia, and of any of the physical signs of

* See a case by M. Valleix, *Archives Générales de Médecine*, vol. xxii., 1850.

† *Vide* Case 137.

‡ *Vide* a case by Hope, *opus citat.*, p. 447; also one by Mayne, *Proceedings of Pathological Society of Dublin*, June 24th, 1873; and another by Bellingham (case 12), *loco citat.*

§ *British Medical Journal*, March 6th, 1875.

aneurism, cough of this character, associated with shooting pains in the chest, has led to the diagnosis of aneurism which was subsequently verified by dissection. The aneurism was found to have arisen from the transverse portion of the arch, and pressed upon the left bronchus, into which it ultimately opened.*

Doctor Stokes mentions a case seen by him in consultation with the late Sir P. Crampton, in which the only symptom produced by a large aneurism of the ascending and transverse portions of the arch of the aorta, was a single sonorous inspiration, like that of whooping cough, at the termination of a fit of coughing.†

Recurrent hæmoptysis, in the absence of laryngeal, pulmonary, and cardiac disease, is likewise eminently suggestive of thoracic aneurism. It may occur by the "leakage" of an aneurism into the air passages, or it may be the result of congestion from the pressure of an aneurism upon the lung-structure, or the pulmonary veins; or from the irruption of an aneurism into the pulmonary artery. When hæmoptysis is produced by the direct communication of an aneurism with the bronchial system, the sputa are either streaked with, or entirely composed of, arterial blood. Pulmonary apoplexy, by aspiration of blood into the air passages, and evinced by the signs of solidification more or less extensive, may complicate an accident of this kind, or it may arise from pressure and local congestion, followed by rupture of the distended vessels. In the latter event, passive pneumonia, attended with the usual physical signs, will follow unless death occur within a short period. The sputa from pulmonary congestion exhibit a uniform intermixture of blood.

Doctor Gairdner holds that hæmoptysis, accompanied by laryngeal stridor not due to ulceration of the larynx, and in the absence of pulmonary disease or solidification, can arise only from a leaking aneurism.‡ This proposition cannot be disputed. In such cases, however, other symptoms and certain signs of aneurism, are usually exhibited, especially laryngeal cough and inequality of respiration.

* O'Ferrall, *Proceedings of Pathological Society of Dublin*, March 24th, 1860.

† *Opus citat.*, p. 559.

‡ *Clinical Medicine*.

Doctor A. Fauvre regards visible pulsation with tortuosity of the subclavian arteries, accompanied by tremor and double murmur, as indicative of aneurismal dilatation of the superior wall of the transverse portion of the arch. The inflection of the subclavian arteries, whence the phenomena in question arise, is due, he thinks, to loss of elasticity and consequent elevation of the superior wall of the aorta.* I have met with an example of this kind in the person of a female of middle age and a highly nervous temperament. The carotid and subclavian arteries were remarkably tortuous, and under the slightest emotional excitement they pulsated with great violence. Immediately above the sternal end of the right clavicle, the common carotid was sharply bent upon itself, and formed a visible tumor which yielded a throbbing pulsation closely simulating that of innominate aneurism.

The existence of an aneurism of the arch of the aorta may be entirely masked, in regard both to symptoms and signs. Such examples are designated *latent*. In exceptional instances, large tubular aneurisms involving the entire arch may be unattended by symptoms or signs of any kind, except neuralgic pains diffused over the chest, shoulders, and neck, and an accentuated second sound in the aorta.† Small aneurisms arising from the left extremity of the arch, and involved in the substance of the left lung, are, however, those which are most frequently latent. The existence of such has been, in many cases, announced only by the occurrence of a fatal hæmorrhage. Several examples of this kind have been exhibited before the Pathological Society of Dublin by Drs. Mayne, O'Ferrall, Lees, Hudson, Banks, and Symes.‡

With regard to the use of the sphygmograph in the diagnosis of thoracic aneurism, Mr. Mahomed declares that, "If the aneurism is one of an artery going to the upper extremity, and the aorta be not involved, valuable evidence will be afforded :

"If the aneurism is one of a similar artery, involving the aorta, frequently no difference can be detected in the pulsation of the radials.

* *Archives Générales de Médecine*, in *London Medical Record*, June 10th, 1874.

† *Vide Case 139.*

‡ *Vide Proceedings, passim.*

"If the aneurism be one of the aorta itself, and situated in the ascending part of the arch, no difference in the pulse of either side can be detected.

"If, of the transverse part of the arch, the pulse may or may not give evidence of its existence."*

Aneurism of the arteria innominata may be now briefly considered, chiefly in reference to the differential diagnosis from aneurism of the aorta.

This subject has been treated exhaustively by Dr. Holland,† who has collected and epitomised from various sources 46 recorded examples of the disease. Innominate, as contrasted with aortic aneurism, presents the following distinctive peculiarities, as deduced by Dr. Holland from an analysis of these 46 cases. (a) More frequent appearance of external tumor; (b) more frequent dislocation of the larynx and trachea; (c) more frequent congestion of the right side; (d) less frequent occurrence of diffused or radiating pain in the chest; (e) more frequent occurrence of pain in the right side of the head and neck, and the right arm; (f) more frequent occurrence of cedema and paralysis confined to the right arm; (g) more frequent occurrence of diminished pulsation in the arteries of the right side of the neck and right arm; and (h) the decided effect of pressure upon the right carotid and subclavian artery in diminishing and arresting the pulsation of the sac.

Amongst the distinctive peculiarities of innominate aneurism, the seat of pulsation and sound behind the right sterno-clavicular articulation, the early appearance of a tumor above the clavicle, and of the signs of vascular obstruction on the right side, and the frequent displacement of the trachea and larynx to the left, deserve special notice. It may be further observed that, whilst the intrinsic signs are identical with those of aneurism of the arch, the signs of pressure upon the right bronchus and lung are less frequently exhibited than in the last mentioned disease.

Diminished force of circulation in the right carotid and subclavian arteries at an early period of the disease, is the most constant symptom of innominate aneurism. Dr. Sibson has

* *Medical Times and Gazette*, August 30th, 1873.

† *Dublin Quarterly Journal of Medical Science*, vol. xiii., 1852.

calculated that the former vessel is obstructed in one-half, and the latter in nine-tenths of the cases.* The aorta is frequently involved in aneurism of the arteria innominata. Towards the determination of this point, which is of the utmost importance in regard to surgical treatment, but in many cases attended with the greatest difficulty, the sphygmograph is capable of affording invaluable assistance. According to Mahomed, the exhibition of decided aneurismal characters in the pulse-tracing of the right side, might be deemed conclusive as *against* the supposition that the aorta was involved.

Doctor Stokes adverts to the singular variation of the voice, "from the deepest bass to a shrill treble," exhibited in a case of innominate aneurism which came under his notice.†

In the progress of growth, an aneurism of the innominate artery may transgress the middle line, and invade the parts on the left side of the neck, as occurred in a case reported by the late Dr. Hutton.‡ In a case published by Dr. McDowel, the greater portion of the tumor lay within the chest, where the physical evidence of its presence was masked by the intervention of the right lung between the tumor and the anterior wall of the chest.§ In this case, and in Dr. Hutton's, both of which were diagnosed at an early period, the first symptom exhibited was severe pain in the right side of the neck, the right shoulder, and ear. Recurrent laryngeal spasm, accompanied by paroxysms of dyspnoea from tension of the right recurrent nerve, may likewise result from the growth of an aneurism of the innominata. An example of this kind has been published by Dr. Hughes Bennett,|| and another by Dr. Gairdner.¶

The immediate *cause of death* and the mode of its occurrence in thoracic aneurism, are various. Death may be caused by intercurrent disease, acute or chronic, or by disease directly or remotely consecutive to the aneurism; as bronchitis from local

* Croonian Lectures, *Lancet*, April 9th, 1870.

† *Dublin Journal of Medical Science*, vol. v.

‡ *Ibid.*, vol. xxv.

§ *Proceedings of the Pathological Society of Dublin*, vol. iv., part i.

|| *The Principles and Practice of Medicine*, second edition, p. 563.

¶ *Clinical Medicine*, p. 470.

irritation, congestive pneumonia or gangrene of the lung, atrophic softening or abscess of the brain, or by phthisis. An intimate association between aneurism and pulmonary tuberculosis has been alleged by Dr. Stokes, as a legitimate inference from clinical observation. I have met with one case (No. 142) which appeared to afford very decided evidence in support of this doctrine. The patient, a man aged fifty-four, with no hereditary predisposition, and no constitutional tendency to phthisis, died of aneurism of the ascending and transverse portions of the arch of the aorta, after an illness of thirteen weeks' duration, in the course of which no hectic symptoms were exhibited. Both lungs were studded with recent miliary tubercle. Several cases in which aneurism and pulmonary tubercle coexisted have been published in the *Proceedings of the Pathological Society of Dublin*. Rokitsansky insists on a positive antagonism between aneurismal and the tuberculous diathesis.* Dr. Walshe has met with only 2 examples of pulmonary tuberculosis in 16 cases.† The doctrine of affinity between aneurism and tubercle must, therefore, though probable, be regarded as still unproven.

With regard to the mode in which death has occurred, directly from thoracic aneurism, the 68 cases given in Table XVI. yield the following results: Death occurred by exhaustion from pain and insomnia in 11 instances; from repeated small hæmorrhages in 1; asphyxia 9; asthenia the result of mal-nutrition 5; syncope 5; coma 4; bronchitis 4; pleuro-pneumonia 1; and from rupture of the aneurism in 26 instances. Rupture into the pericardium occurred in 10 cases; into the trachea in 4; right pleura 3; left lung and pleura 3; left bronchus 2; left lung 1; left pleura 1; left bronchus and cesophagus 1; and externally 1. Rupture into the left lung or pleura was more frequent than into the right in the proportion of 8 : 3. As judged by the preceding results, then, death from thoracic aneurism is most frequently due to rupture of the sac. It results from other causes in the following order of frequency: exhaustion or debility, acute pulmonary inflammation, and coma.

Hæmorrhage from rupture of a thoracic aneurism may be

* *Pathological Anatomy*, vol. i., p. 315.

† *Diseases of the Heart*, fourth edition, p. 499.

rapidly fatal by syncope, or by asphyxia; or slowly by asthenia from repeated small bleedings.

Fatal syncope by loss of blood may result from a single and copious hæmorrhage, externally, into either pleura, the œsophagus, or the stomach; or by compression of the heart from the escape of a few ounces of blood into the pericardium.*

The opening of an aneurism into the trachea or any portion of the bronchial system may be followed by death from asphyxia within a few minutes. The aperture may, however, be plugged by fibrin, and death postponed for an indefinite period.†

Hæmorrhage, by rupture of an aneurism upon the cutaneous surface, is necessarily preceded by visible tumor and slough, and is, therefore, never unexpected. On the detachment of the slough the first gush from the sac may be fatal, but this is rarely the case. It usually happens that, after the loss of a few ounces of blood, syncope supervenes, and hæmorrhage is arrested owing to the temporary debility of the heart and the formation of a plug of fibrin in the aperture. This object may be likewise effected by mechanical means.

Doctor Stokes mentions a case under the care of the late Dr. Osborne, in which an external opening by detachment of slough in an aneurism of the chest, was closed at each systole by a plug of fibrin; the plug receded with each diastole. It was at length dislodged, and a formidable gush of blood followed, but further hæmorrhage was stayed by the nurse, who plugged the opening with a cotton apron. By means of this *tampon* the patient's life was prolonged for several days.‡ In the course of the session 1864-5, Dr. Minchin reported to the Pathological Society a somewhat similar case.§ A large aneurism of the transverse portion of the arch of the aorta had pointed in the neck, and sloughed at its most prominent point. A slight oozing of blood occurred by filtration through the sloughing integument; this

* Both Laennec and Hope declared they had never met with an example of rupture of an aneurism into the pericardium.

† Dr. Gairdner has recorded a case in which an opening into the trachea became blocked by a mass of fibrin, and death did not occur till four years afterwards. Mr. Liston lived five months after an opening into the trachea had been formed.

‡ *Opus citat.*, p. 580.

§ Vol. ii., part iii., p. 192, No. 29, Table XVI.

continued for eight days. On the ninth day, an opening as large as a sixpenny piece was formed by detachment of a slough. This was, however, plugged by a mass of fibrin, which accurately closed the opening during the systole of the ventricle, but permitted a slight escape of blood during diastole. On the tenth day the opening had attained the size of a half-crown. In this opening a large globular mass of fibrin, which accurately fitted it, was seen to move alternately forwards and backwards with the systole and diastole of the ventricles. On the eleventh day from the first occurrence of hæmorrhage, this plug, which was as large as a man's fist, was suddenly shot out of the opening, followed by a gush of blood proving instantly fatal.

A still more remarkable example of protracted external hæmorrhage from an aneurism of the aorta, is mentioned by Dr. Stokes.* The patient, a man aged fifty-six, under the care of the late Dr. Neligan in the Jervis-street Hospital, presented a large aneurism in the right mammary region. It had passed through the chest-wall, and it yielded a strong impulse and a double murmur; the voice was husky, and there was paroxysmal cough with dyspnœa. This tumor had existed for a year, and continued, during that period, to discharge blood in small quantity through a sinuous opening on its outer surface. Repeated hæmorrhage, in jets, occurred under Dr. Neligan's observation, but was easily restrained by mechanical means. Under a nutritious diet the tumor became solid, the pulsation diminished, cough and dyspnœa ceased, and the sinus entirely closed. The patient ultimately left hospital, declaring he felt quite well.

A case observed by the late Professor Smith is mentioned by the same writer, in which an external hæmorrhage from an aneurism of the aorta that had perforated the sternum, was restrained for ten days by a compress and roller. The patient ultimately died of exhaustion from continued leakage through the opening.†

A case, in which external hæmorrhage from an ulcerated opening in a large aneurism of the arch of the aorta was restrained for sixteen days by a plug of fibrin, has been published by Dr. W. H. Webb from the clinique of Dr. Da Costa. Dr. Webb gives

* *Opus citat.*, p. 582.

† *Opus citat.*, p. 581.

a list of seventeen cases of aneurism of the thoracic aorta, in which external hæmorrhage by rupture of the sac had occurred. The duration of life after the first hæmorrhage varied in these cases from seven weeks to a few seconds.*

Serous membranes usually give way suddenly, and by rent, under the tension of an aneurism.

Hæmorrhage by rupture into either pleural cavity is usually fatal on the instant. Such, however, is not necessarily the case. As on the external surface of the body, hæmorrhage into a serous cavity may take place "by successive gushes, between the periods of which complete reaction may occur," as truly remarked by Dr. Stokes.

Laennec maintains that the left pleura is, more frequently than the right, the seat of hæmorrhage from rupture of a thoracic aneurism. In this opinion, which is borne out by the statistics furnished in Table XVI., I entirely concur.

Thurnam avers that the occurrence of a consecutive false aneurism of the intra-pericardial portion of the aorta is not possible, owing to the absence of an external or areolar coat in this portion of the vessel.† Hence, aneurisms within the pericardium usually prove fatal at an early period of their growth, and by simultaneous rupture of the sac and its serous investment. But the frequent occurrence of dissecting aneurism in this situation proves that the rule in question admits of many exceptions. Even the actual occurrence of hæmorrhage into the pericardium is not necessarily fatal on the instant. In the case of the late Dr. Ball, reported by Dr. Stokes, the patient survived, for one hundred and eight hours, the rupture of an aneurism into the pericardium by two small rents in the serous membrane.‡ In a case recorded by Dr. Finny, hæmorrhage into the pericardium, by rupture of an aneurism of the aorta immediately above the valves, was restrained by adhesion of the pericardium to the heart. Death did not occur till twenty-four hours after rupture took place, as indicated by the symptoms.§

* *American Journal of the Medical Sciences*, No. 136, new series, October, 1874.

† *Medico-Chirurgical Transactions*, vol. xxi., p. 232.

‡ *Proceedings of the Pathological Society of Dublin*, April 25th, 1857.

§ *Ibid.*, vol. iii., part ii., new series; see also a case by Dr. Law, *Dublin Journal of Medical Science*, vol. xxi.

Hæmorrhage into the superficial structures of the chest by subcutaneous rupture of the sac, constituting secondary false aneurism, is seldom fatal for many days after its occurrence. The intrinsic signs are, however, in a great degree modified by such an event. A case has been recorded by Dr. Bellingham in which a thoracic aneurism, pointing externally, and characterized by a double impulse and a double sound, became diffused under the pectoral muscles. Of the previous signs, systolic impulse alone remained, but it was now accompanied by murmur.*

Death from thoracic aneurism may occur by cerebral congestion and coma, as when the sac opens into the superior vena cava, or occludes it by compression; by spinal paralysis consequent upon rupture of the sac into the spinal canal; by sudden thrombosis of the aorta in connexion with a similar condition of the left ventricle;† or slowly, by inanition from compression of the œsophagus.

The *treatment* of thoracic aneurism should have reference to alleviation, and to cure. The paroxysmal and neuralgic pain of aneurism is promptly mitigated or suspended by the hypodermic use of morphia. One-sixth to one-fourth of a grain of the acetate or hydrochlorate of morphia, in aqueous solution, injected under the skin of the arm by means of the ordinary hypodermic syringe, rarely fails to bring, within a few seconds, partial or complete relief from pain, which lasts from two to six hours.‡ Opium, given in pill or in draught, is less efficacious. If the pain proceed from tension of the sac, as indicated by a feeling of oppression and constriction of the chest, with superficial hyperæmia and tenderness in the seat of tumor, local abstraction of blood by means of a few leeches, followed by a warm poultice, will give immediate relief. In cases of general or local venous congestion from pressure upon one of the principal veins, or of great oppression of breathing with hæmoptysis, bleeding from the arm, by reducing the volume of the blood and lowering vascular

* *Dublin Medical Press*, April 19th, 1848.

† *Vide* case by Dr. J. S. Hughes, in *The Medical Press and Circular*, Oct. 15th, 1873.

‡ I have latterly used the compound solution of sulphate of morphia and atropia recommended by Dr. James Little (*Dublin Journal of Medical Science*, vol. liv.) m v of this solution contains $\frac{1}{2}$ of a grain of morphia, and $\frac{1}{135}$ of a grain of atropia.

tension, will be found more efficacious than local abstraction. The bleeding should be repeated from time to time, and the quantity should be regulated according to the degree of congestion and the urgency of dyspnoea. I think there are few cases in which, even in the advanced stages of aneurism, the patient will not, in such circumstances, derive benefit from the loss of six to eight ounces of blood. In these cases, likewise, and with the same object in view, hydragogue aperients should be occasionally administered, whilst the allowance of liquids to be drunk should be restricted as far as practicable. For the purpose of insuring sleep, opium, chloral hydrate, or bromide of potassium, should be given in adequate doses, if necessary. I have found a combination of the two latter, in doses of xv grs. to xx grs. of each, more efficacious than either given alone.

The curative treatment of aneurism should be directed to the twofold object of effecting closure of the sac by deposition of fibrin, and arresting further arterial change by promoting general nutrition. In the system of Valsalva and Albertini the latter object was lost sight of. Hence, irrespectively of its irksomeness, and the consequent difficulty of pursuing it for a sufficient length of time, this system of treatment, which consisted in copious and repeated bleeding from the arm, active purgation, and a starvation diet, with absolute rest in the recumbent posture, was defective even in its conception.* M. Bertin was the first writer who took exception to this system, and ventured to question the success claimed for it in the treatment of aneurism of the aorta. He adopted the modified plan of leeching, enjoining absolute repose of body and mind, and restricting within moderate limits the diet of the patient.†

Doctor Proudfoot, in the same year (1824), published two cases, in the treatment of which leeching and purgation were the only portions of Valsalva's system adopted.‡

Doctors Graves and Stokes, whilst recommending this system of treatment in the early stages of thoracic aneurism, suggested, in all other cases, the opposite plan in regard to diet, which had been

* *Vide Morgagni de Sedibus et Causis Morborum*, lib. ii., epist. xvii., § 30 et 31.

† *Traité des Maladies du Cœur*, p. 151.

‡ *Edinburgh Medical and Surgical Journal*, vol. xxii.

found more satisfactory by Proudfoot and Beatty.* Dr. Stokes has more recently objected to a lowering plan of treatment on several grounds: The advanced age of most of the subjects of aneurism; and the feeble state of nutrition, the irritability of the heart, and the hypinotic state of the blood produced by it.

With the view of promoting a deposition of stratified fibrin within the sac, and its subsequent solidification, measures must be taken to ensure cardiac and vascular quiescence as the first condition of success. All other means are subordinate to these. Hence, "Tufnell's plan," under which the patient is strictly confined to the recumbent posture for two or three months, supplies the most essential condition of a rational system of treatment.

According to Bellingham, to whom the merit of conceiving this plan is due, and Tufnell, who has developed and applied it with considerable success, the patient's diet and drink must be likewise restricted within prescribed limits.

Bellingham would limit the patient to three meals a-day: the morning and evening meal to consist of two ounces of liquid and four of solid nutriment; the mid-day meal, of four ounces of liquid, with from four to six ounces of solid. The liquid may consist of milk or tea, the solid of bread, and the mid-day meal of bread and meat in equal quantity.† This dietary must be strictly persevered in for a month or six weeks, whilst repose of mind and body is, as far as possible, to be maintained. The bowels should be kept free, but systematic purgation is deprecated.

Mr. Tufnell has, with considerable success, pursued this plan of treatment in a still more rigid form.‡ The patient is strictly confined to the horizontal posture for a period varying from eight to thirteen weeks, according to the effect upon the aneurism. During this period, the only change of posture permitted is turning slowly upon either side, or upon the face, whilst, by special arrangement of the bed, the bowels and bladder may be evacuated without disturbance of the body. The following scale of diet is, as far as practicable, to be carried out, viz.: For breakfast, two ounces of white bread and butter, with two ounces of cocoa or milk; for dinner, three ounces of meat, with three

* *Dublin Hospital Reports*, vol. v., 1830.

† *A Treatise on the Diseases of the Heart*, 1857, p. 620.

‡ *The Successful Treatment of Internal Aneurism*, 1864.

ounces of potatoes or bread, and four ounces of water or light claret; for supper, two ounces of bread and butter, and two ounces of milk or tea; the total amount of solid food allowed in the twenty-four hours being ten ounces, and of liquid food, eight ounces. After the contents of the sac have been in some degree solidified, or earlier if the patient prove restive under restriction, a more liberal scale of diet is allowed. As to medicine, sedatives, and especially opium, are given according to the urgency of pain, whilst moderate relaxation of the bowels is to be effected by means of ordinary aperients.

Mr. Tufnell has reported six examples of solidification of aortic aneurism under the foregoing plan of treatment, viz.: two of thoracic, and four of abdominal aneurism.

In a recent communication Mr. Tufnell has adduced, from his own practice, three additional examples of the successful treatment of aneurism by the same means, viz.: two of aneurism of the abdominal aorta, which were solidified in thirty-seven and twenty-one days, respectively; and one of popliteal aneurism cured in twelve days.*

The importance of the principle involved in this mode of treating internal aneurism, and the encouraging results obtained from its adoption, must be my apology for the foregoing lengthened notice.

As an element in the curative treatment of aneurism, general blood-letting is likewise occasionally demanded. Vascular plethora and extreme tension of the sac may be most promptly corrected by bleeding to the amount of ten to twelve ounces, and should be practised where not absolutely forbidden by the debility of the patient. Dr. D. Gatzuc has shown experimentally that the withdrawal of blood from a vein or artery retards the current and reduces the blood-pressure, and in proportion to the quantity drawn.† Therefore, whether for the purpose of averting rupture of the sac, promoting deposition of fibrin within it, or of alleviating urgent symptoms arising from excentric pressure, an occasional bleeding by venesection may be resorted to with advantage in suitable cases.

Where this seems inadmissible, the object may be accom-

* *Medico-Chirurgical Transactions*, vol. xxxix., second series, 1874.

† *British and Foreign Medico-Chirurgical Review*, April, 1872.

plished by the application of six to twelve leeches at the point where the sac is nearest to the surface. But should the aneurism project externally, the leeches should be applied, not over the tumor, but immediately around it.

The medicinal agents which have been used with the object of effecting or promoting fibrination in the sac, may be now briefly noticed.

Acetate of lead has been strongly recommended by Hope. An example of success by means of this agent, given in doses of grs. iv to viii, thrice daily, has been reported by Hoegh.* Some doubt is, however, expressed by the author as to the accuracy of his diagnosis. Mr. T. Holmes has judiciously remarked that painters are notoriously prone to aneurism. I have given the acetate of lead a fair trial, but without apparent benefit.

Iodide of potassium has been long used in the treatment of aneurism. According to Dr. Walshe, it was first recommended by Bouillaud in 1859, and next by Chucurbatty in 1862. In 1869, Dr. George W. Balfour recorded several cases of aneurism successfully treated with the iodide,† and still more recently two additional examples of aneurism, cured by means of large doses of this drug, were reported by the same physician.‡ The aneurism in one of these cases was thoracic, and was cured after a treatment of twenty months. In the second case, both an innominate and an abdominal aneurism existed. The doses of the iodide given, varied from grs. x to grs. xv, thrice daily. A case of thoracic aneurism, successfully treated with the same agent, has been published by Dr. Dyce Duckworth.§

My own experience of the iodide of potassium in the treatment of aneurism is rather favourable. I have tried it in several cases in the doses recommended by Dr. Balfour; but in no instance have I given it for more than one month. I have found it to relieve pain in some instances, an effect which I attribute to its property of lowering blood-pressure, and so lessening the tension of the sac.

* *Medical Press and Circular*, June 23rd, 1869, translated by W. D. Moore from *The Norsk Magazin*, 1868.

† *Edinburgh Medical Journal*, 1869.

‡ *Ibid.*, December, 1871.

§ *British Medical Journal*, August 23rd, 1873.

Ergotin has been used hypodermically by Langenbeck, with a view of solidifying the contents of the sac.*

I have tried phosphorus, with the object of arresting vascular degeneration, but only in one case (No. 142), and in that instance the disease was too advanced to admit a hope of much benefit from medicine of any kind. At an earlier period of the disease it may, perhaps, be found useful.

Galvano-puncture has been used by several physicians: Ciniselli,† McCaul Anderson,‡ and Charlton Bastian.§ Complete success has attended this plan of treatment in the hands of Ciniselli and Dr. Anderson. Out of 12 cases in the practice of Ciniselli, 10 were reported as well within periods varying from twelve to twenty-one months after operation; relapse occurred in 2 of the cases, in one of these a second and successful operation was performed, the other died within eighteen months; death resulted directly from the operation in 4 cases. Another successful case by Ciniselli has been reported by Dr. Althaus.|| In Dr. Anderson's case, four operations were performed. The patient, though not perfectly cured, was reported as well seven months afterwards.

Mr. C. H. Moore and Dr. Murchison made the bold attempt to bring about coagulation by introducing several yards of iron wire into the sac. Death occurred on the fifth day after operation.¶

Where the tumor points externally and the sac is making progress towards the surface, a well-adjusted pad, strapped down over it by means of adhesive plaster, will not only relieve the pain arising from the tension of the skin, but will likewise retard the advance of the aneurism.

More than a reference to the surgical treatment of aneurism of the aorta by distal pressure or ligature, would manifestly be out of place in this work. For a full discussion of this subject, the reader is referred to the admirable and exhaustive lectures of Mr. Timothy Holmes.**

* *Berlin. Klin. Woch.*, No. 12, 1869; Abstract in *Dublin Journal*, May, 1870.

† Quoted by Mr. Holmes, *Lancet*, November 9th, 1872.

‡ *Lancet*, vol. i., 1873.

§ *British Medical Journal*, November 22nd, 1873.

|| *Treatise on Medical Electricity*.

¶ *Medico-Chirurgical Transactions*, vol. xlvii., 1864.

** *On the Surgical Treatment of Aneurism*, *Lancet*, July 13th and 27th, 1872.

A remarkable example of success from the distal ligature has been reported by Mr. C. Heath.* The case was one of aneurism involving the ascending and transverse portions of the arch of the aorta, and pressing on the left subclavian artery. A ligature was applied to the left carotid above the omo-hyoid muscle. The tumor receded, and all the previous signs disappeared. A year after the operation, the patient, a man aged forty-eight, was reported as having been, in the interim, actively employed as a farm-labourer. The prominence of the chest in the seat of tumor had then disappeared; dulness was gradually clearing up, and the man was virtually in perfect health.

Another successful case has been reported from the practice of the same surgeon.† In this case, which was one of aneurism of the arch of the aorta involving the origin of the arteria innominate, both the carotid and the subclavian artery of the right side were ligatured. The patient, a woman, left hospital convalescent three months afterwards; but, owing to the administration of an emetic, the aneurism again became active, and soon proved fatal.

A case in which the distal ligature was used for the cure of an innominate aneurism has been recorded by the late Mr. Hutton. Secondary hæmorrhage occurred, and the patient died by syncope on the seventy-sixth day after the operation.‡

As to the use of distal compression for the cure of an innominate aneurism, under the prolonged administration of chloroform, Mahomed declares that this procedure is not entirely free from danger. Amongst the consequences to be apprehended are apoplexy, of which he gives an example from compression of the right carotid for the cure of an aorto-innominate aneurism; retroversion of a diseased aortic valve; dilatation of the left ventricle, with aggravation of pre-existing mitral reflux; and rupture of the sac. Where left ventricular hypertrophy and general atheroma exist, compression, if attempted, should be made without chloroform, or a ligature should be applied.§

The following Table (XVI.) has been constructed from the records of the Pathological Society of Dublin.

* *Clinical Society's Transactions*, vols. v. and vi.

† *British Medical Journal*, May 21st, 1870.

‡ *Proceedings of the Pathological Society of Dublin*, December 10th, 1842.

§ *Medical Times and Gazette*, August 9th, 1878.

TABLE XVI.]

ANEURISM OF THE AORTA WITHIN THE THORAX.

No.	Author.	Sex, Age, and Occupation.	Habits, and Cause of Aneurism.	Duration in Months, and Seat of Aneurism (where stated).	General Nutrition.	Aneur. Murmur.	Aneur. Sounds.	Aneur. Impulses.	Modes of Death.
1	Dr. Law	Male, 43	..	24: Thoracic aorta	Good	Systolic	None	Systolic	Exhaustion
2	Dr. Smith	Male, 47	..	24: Transv. portion of arch	..	Systolic	None	None	Coma
3	Dr. Law	Male, 42	..	4: Ascend. portion of arch	Coma
4	Dr. Lees	Female, 60	..	Ascend. portion of arch	Rupture into pericard.
5	Dr. O'Ferrall	Male, 45	..	Middle of transv. portion of arch	..	None	None	None	Asphyxia
6	Dr. J. C. Ferguson	Male	..	Ascend. portion of arch	Rupture into pericard.
7	Dr. Bigger	Soldier	..	Ascend. and part of transv. portion of arch	..	None	None	None	Exhaustion
8	Dr. Greene	Male, 39	..	Ascend. portion of arch and abdominal aorta	Emaciated	Systol. in abdomen	None	Systolic	Exhaustion
9	Dr. Lees	Female, 62	..	Transv. portion of arch	Rupt. into left lung
10	Dr. Stokes	Male, 39	Temperate	24: Upper part of descend. aorta	..	Systolic	None	Systolic	Intercurr. bronchitis
11	Sir P. Cramp-ton	Male	..	Junction of ascend. and transv. portion of arch	..	None	Systolic	Systolic	Syncope
12	Dr. Law	Slater, 92	Intemperate, fall from scaffolding	Left extremity of transverse portion	..	None	None	Systolic	Rupt. into left lung & pleura
13	Dr. McDowel	Male, 40	..	Transv. portion of arch	..	None	Systol. and diastol.	Systolic	Asphyxia
14	Sir D. Corrigan	Male, 32	..	2: Upper part of ascend. and whole of transv. portion of arch	..	None	None	None	Rupt. into trachea
15	Dr. O'Ferrall	Soldier, 36	Intemperate	Transv. portion of arch	Emaciated	None	None	None	Rupt. into left bronchus
16	Dr. McDowel	Porter, 35	..	10 days: Transv. portion of arch, engaging origin of arteria innominata	..	None	None	None	Asphyxia
17	Dr. J. F. Duncan	Bricklayer, 40	Intemperate	4: Ascend. portion of arch, involving arteria innominata	..	Systolic	None	Systolic	Coma
18	Dr. Stokes	Porter, 50	..	12: Left extremity of transv. portion	Good	None	None	None	Rupt. into trachea
19	Dr. Mayne	Horse-dealer	Intemperate; kick from a horse on chest	9: Com. of descend. portion of arch	..	None	None	None	Exhaust. by repeated hæmorrhages
20	Dr. Hudson	Male, 34	..	42: Middle of thoracic aorta	..	None	None	None	Rupture into left lung, and pleura
21	Dr. H. Kennedy	Male, 44	..	4: Upper extremity of ascending portion of arch	..	None	None	Systolic	Exhaustion
22	Mr. Symes	Police Constable	..	Left extrem. of transverse portion of arch	Rupture into left lung, and thence into left pleura
23	Dr. Mayne	Compositor, 42	Intemperate	24: Inferior portion of thoracic aorta	..	Systolic	None	Systolic	Asphyxia
24	Dr. Gordon	Porter, 34	..	4: Anterior sinus of Valsalva	..	Systolic	None	None	Asphyxia

No.	Direction of Growth.	Disease of Valves.	State of Ventricles.	State of Aorta.	Observations. General State of Heart.
1	Back. & laterally	None	..	Atheromat. in seat of aneurism	Six aneurisms.
2	Backward	None
3	Back. and to right	Aneurism occluded superior vena cava.
4	Up. & down. in course of aorta	Aortic and mitral	R. Normal L. Hypert. and dilated	Atheromatous	Hypertrophy and dilatation; dissecting aneurism; no previous history.
5	Backward	No dulness, impulse, murmur, or interruption of circulation; paroxysmal dyspnoea, bordering on asphyxia.
6	Backward and downward	Pressure on superior cava, and lividity of face and neck.
7	General expansion	Aortic	R. Dilated L. Dilated and hypertrophied	Atheromatous	Enlarged.
8	Forward in both	Aortic	..	Atheromatous	Double aneurism.
9	Forward, and to left	Atheromatous	No suspicion of aneurism; rupture into substance of left lung at apex, which was attached to sac.
10	Forward, and to right	Symptoms of aneurism well pronounced; also physical signs in upper left front. Both disappeared—former returning after six months; latter not returning.
11	Forward	None	Normal	Healthy except at seat of aneurism	Not hypertrophied. Effusion into pericardium.
12	Back. and to left	None	..	Atheromat. in seat of aneurism	Rupture into apex of left lung, followed by fatal hæmoptysis.
13	Back. and to right	None	Normal	..	Heart fatty; no stridor; asthenia.
14	Back. and to right	None	Normal	Atheromat. in seat of aneurism	No hypertrophy.
15	Back. and to left	No physical sign of aneurism, except loud ringing cough.
16	Down. back. & to left	None	Normal	Atheromatous	Not hypertrophied; fatty; no brist. impulse, or laryngeal signs, but intense paroxysmal dyspnoea.
17	Forward and to right	Aortic	..	Atheromatous	Pressed on superior cava, causing cyanosis and oedema of upper half of body, and contraction of right pupil.
18	Back. and to right	None	Normal	Atheromatous	No hypertrophy; stridor, dyspnoea, or physical signs.
19	Forward and to left	No symptoms or signs. Aneurism was imbedded in apex of left lung, into which it had leaked.
20	Forward and to left	None	No special symptoms or signs. Fixed pain in back, and shooting pains through chest.
21	Forward and upward	None	Normal	Atheromat. in seat of aneurism	No hypertrophy. Symptoms were dysphagia, cough, a pulsating tumor in root of neck, and turgescence of superficial veins of chest.
22	Forward and to left	Atheromatous	Tumor involved in left lung. No suspicion of aneurism.
23	Forward and to left	Slight thick. of aortic valves, which were competent	..	Atheromat. in seat of aneurism	Not hypertrophied. Aneurism involved in left lung, into which it had leaked.
24	Downward and to left	Aortic ruptured	R. Dilated L. Dil. and hypertrophied	Atheromatous	Hypertrophy. Aneurism projecting into right ventricle, and opening into left ruptured ventricle through aortic valve.

TABLE XVI.—*Con.* ANEURISM OF THE AORTA WITHIN THE THORAX.

No.	Author.	Sex, Age, and Occupation.	Habits, and Cause of Aneurism.	Duration in Months, and Seat of Aneurism (where stated).	General Nutrition.	Aneur. Murmur.	Aneur. Sounds.	Aneur. Impulse.	Modes of Death.
25	Dr. Gordon	Male	..	48: Two, viz.; in com. of ascending portion of arch & of thoracic aorta	..	Systolic	None	Systolic	Exhaustion
26	Dr. Banks	Whitesmith, 50	Temperate; severe anvil work	90: Upper portion of ascend. aorta	..	None	Two	Systolic and diastolic	Exhaustion
27	Mr. Symes	Male, 50	..	Entire extent of aorta
28	Dr. Banks	Male, 46	..	86: Left extremity of transverse portion of arch	..	None	Two	Systolic and diastolic	Syncope
29	Dr. Minchin	Female, 44	..	11: Middle of transv. portion of arch	..	Systolic	None	Systolic	Rupture externally
30	Dr. E. Hamilton	Police Constable	Severe duty	Entire of transv. portion of arch	Emaciated	None	None	Systolic	Rupture into trachea
31	Dr. Jennings	Dischgd. Soldier, 36	..	36: One of the sinusses of Valsalva	..	None	None	None	Exhaustion
32	Dr. Banks	Soldier, 28	..	Com. of transverse portion of arch	Good	None	None	None	Rupture into left pleura
33	Mr. Porter	Male, 68	..	24: Descending portion of arch	..	None	None	None	Asthma
34	Dr. E. Bennett	Coal-porter, 40	Intemperate	One of the sinusses of Valsalva	..	Systolic	None	Systolic	Bronchitis
35	Dr. Banks	Shoemaker, 46	Temperate	6: Whole of transv. portion of arch	Worn	None	Two	Systolic	Syncope
36	Dr. Finny	Soldier	Temperate	One of the sinusses of Valsalva	Good	Systolic	None	Systolic	Rupture into pericard.
37	Dr. Jennings	Labourer, 44	Strain	120: Ascending portion of arch	..	None	None	Systolic and diastolic	Exhaustion
38	Dr. Richardson	Female, 40	..	6: Termination of ascend. and whole of transv. portion, including arteria innominata	..	None	None	Systolic	Pneumo-pneumia
39	Mr. Porter	Shoemaker, 37	Intemperate; great physical exertion	Ascending portion of arch.	Good	None	None	None	Rupture into pericard.
40	Dr. H. Kennedy	Female, 60	..	24: Junction of ascend. and transv. portion of arch	..	None	None	Systolic	Asthma
41	Dr. Law	Butler, 60	Intemperate	Entire of ascending portion of arch	Good	None	None	None	Syncope
42	Dr. Stokes	Gentleman 54	Temperate; physical effort	24 hours; ascending portion of arch	Good	None	None	None	Rupture into pericard.
43	Dr. Quinlan	Male, 40	..	Junction of transv. and ascend. portion of arch	..	None	None	None	Syncope
44	Dr. Lyons	Male, 45	Intemperate; blow upon chest	36: Com. of ascend. portion of arch	..	None	None	None	Rupture into pericard.
45	Dr. Lyons	Sailor, 36	..	8: Ascend. portion of arch	..	Diastolic	Systolic	Systolic and diastolic	Rupture into circumscribed cavity in R. pleura.
46	Dr. Lyons	Labourer	..	8 days: two aneurisms, one in up. part of ascending port. of arch, and the other at root of vessel	..	Systolic	None	None	Rupture into pericard.
47	Dr. W. Moore	Cardriver	Intemperate	8: Com. of thoracic aorta	Emaciated	None	None	None	Rupt. into left bronchus
48	Dr. MacSwiney	Male	..	Some months; thoracic aorta	..	None	None	None	Rupt. into right pleura

No.	Direction of Growth.	Disease of Valves.	State of Ventricles.	State of Aorta.	Observations. General State of Heart.
35	The first for. & down., & the second for. & to left	Aortic	..	Atheromatous	Two aneurisms.
36	Forward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied. Constant pain in chest, neck, and arm, with severe paroxysms.
37	Course of vessel	Aortic and mitral	R. Not stated L. Dilated and hypertrophied	Atheromatous	Hypertrophy. Subject sent for dissection. No history; dissecting aneurism from root of aorta to left extremity of iliac artery, into which stream re-entered.
38	Forward and backward	None	Normal	Atheromat. in seat of aneurism	Not hypertro.; contraction of left pupil; great variation in direction of growth.
39	Forward and upward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied; external hæmorrhage by dislodgment of fibrinous plug.
40	Backward	None	Normal	Atheromatous	Not hypertrophied. Occlusion of right subclavian artery.
41	Upward and to right	Aortic and mitral	Dilated and hypertrophied	Atheromatous	Hypertrophied. Advanced valvular disease.
42	Forward and to left	None	Normal	Atheromatous	Not hypertrophied. No suspicion of aneurism, and no symptoms except very slight cough.
43	Atheromatous	Had had popliteal and axillary aneurism.
44	Downward into R. ventricle	None	R. Normal L. Hypertro.	Atheromat. at seat of aneurism	Hypertrophy. Aneurism projected into right ventricle.
45	Forward	Aortic & mitral	Dilated	Atheromatous	Dilated and fatty.
46	Upward	None	R. Normal L. Hypertro.	Healthy	Hypertrophy; aneurism, size of walnut
47	Forward	None	Dilated	Atheromatous	Heart dilated and fatty. Atrophy of brain and recurrent paralysis.
48	General expansion	None	Normal	Atheromatous	No hypertrophy; heart fatty.
49	Forward	Atheromatous in seat of aneurism	Two aneurisms. Man had run a race a few days before death. No suspicion of aneurism.
50	Forward and to right	None	Hypertrophied	Atheromatous	General hypertrophy.
51	Forward and to right	None	Normal	Atheromatous	Not hypertrophied, soft and fatty.
52	Course of vessel	None	Normal	Atheromatous	Not hypertro. Aortic and pulmonic valves atrophied and cribriform; heart fatty; "dissecting" aneurism bursting into pericardium at root of aorta. The patient (Dr. Ball) survived the rupture of internal and middle coats 168 hours; rent in vessel, two-thirds round.
53	Forward and to left	None	..	Atheromat. in seat of aneurism	Hypertrophied.
54	Course of vessel	None	..	Atheromatous	"Dissecting" aneurism. Rent in coats of vessel two-thirds round, probably occurred three years before.
55	Forward	None	..	Atheromat. in seat of aneurism	Double murmur in aorta from inadequacy, the result of dilatation of the vessel.
56	Upper forward, and lower in course of vessel	None	..	Atheromatous	The lower was a "dissecting" aneurism.
57	Forward	None	..	Atheromatous	Aneurism not suspected.
58	Forward and to right	Atheromatous	Heart fatty. No history, as man was moribund when admitted.

TABLE XVI.—*Con.*

ANEURISM OF THE AORTA WITHIN THE THORAX.

No.	Author.	Sex, Age, and Occupation.	Habits, and Cause of Aneurism.	Duration in Months, and Seat of Aneurism (where stated).	General Nutrition.	Aneur. Murmur.	Aneur. Sounds.	Aneur. Impulse.	Mode of Death.
49	Dr. Eames	Male	Temperate	4: Three aneurisms; one from right anterior sinus of Valsalva, and two in ascend. port. of arch above former	..	Systolic	None	None	Rupture of second aneurism into trachea
50	Dr. Law	Labourer, 40	Temperate	4: Posterior sinus of Valsalva	..	Systolic and diastolic	None	None	Coma
51	Mr. J. Hamilton	Pensioner, 39	Strain in lifting weight	8: Transv. port. of arch at middle	..	None	Two	Systolic	Asphyxia
52	Surgeon J. S. Chartres	Soldier, 36	Intemperate; crush by a horse falling on him	24: Com. of thoracic aorta	Emaciated	None	None	None	Exhaustion
53	Dr. McDowel	Poor woman, 65	..	Commencement of ascending portion of arch	Good	None	None	None	Rupture into pericard.
54	Dr. MacSwiney	Poor woman, 40	..	24: Entire arch	..	None	Two	None	Bronchitis
55	Dr. A. W. Foot	Shoemaker, 40	..	8: Com. of ascend. and mid. of transv. portion of arch	..	Systolic in lower aneur.	None	None	Bronchitis
56	Dr. T. B. Little	Poor woman, 38	Intemperate	12: Ascending portion of arch	Good	None	None	Systolic	..
57	Dr. Lyons	Groom, 42	Kick from a horse on the chest	18: Ascend. portion of arch, and lower portion of thoracic aorta	Good	None	Two	Systolic and diastolic	Exhaustion
58	Dr. Finny	Dairyman, 33	Intemperate	12: Ascend. portion of arch	Emaciated	Systolic	None	None	Exhaustion
59	Dr. G. F. Duffey	Soldier, 30	Temperate	30: Ascend. portion of arch above left valve	Good	Rupt. into pericard.
60	Dr. Hayden	Cabdriver, 33	Intemperate; severe blow on back	4: Ascend. portion of arch	Good	None	Two	Systolic and diastolic	Rupture into right pleura
61	Dr. Hayden	Hatter, 34	Intemperate	9: Superior thoracic	Fair	None	None	None	Rupt. into left bronchus and oesophagus
62	Dr. Hayden	Cabdriver, 28	Intemperate; strain in lifting a weight	39: Inferior thoracic	Fair	Systolic	None	Single and systolic	Asthenia
63	Dr. Hayden, per Dr. Stokes	Player upon a wind instrument, 46	..	7 weeks: Ascending portion of arch	Good	Systolic and diastolic	None	None	Slow asphyxia
64	Dr. Hayden	Egg-packer, 33	Intemperate; carrying heavy load	6: Left extremity of arch	Good	None	None	None	Asthenia
65	Dr. Hayden	Architect, 56	Not intemperate	12: Ascend. & transv. portion of arch	Good	None	None	None	Rupt. into pericard.
66	Dr. Hayden	Housekeeper, 31	Temperate	1: Transverse	Good	None	None	None	Asphyxia
67	Dr. Hayden	Mason, 33	Intemperate; strain in lifting a weight	5 weeks: Com. of ascending portion of arch	Good	Systolic	None	Systolic	Asthenia
68	Dr. Hayden	Labourer, 54	Intemperate	5 weeks: Ascending portion of arch	Good	None	Two	Double	Slow asphyxia

No.	Direction of Growth.	Disease of Valves.	State of Ventricles.	State of Aorta.	Observations. General State of Heart.
49	Forward and downward	None	Normal	Atheromatous	Not hypertrophied. Liver depressed by lower aneurism.
50	Downward, forward, and to right	None	R. Dilated L. Dilated and hypertrophied	Atheromatous	Hypertro. and dilat. Depression of liver and jaundice. Aneurism projected into right auricle, and, by blocking vena cava, caused general congestion and dropsy.
51	Forward, upward, and to right	None	Not hypertro. Aneurism had ascended on right side of neck, in course of carotid artery.
52	Forward, and to left	None	Normal	Atheromatous	Not hypertrophied.
53	Course of vessel	.	..	Atheromat. in seat of aneurism	Dissecting aneurism; commenced by rent of internal coat one inch above valves, extended some distance upwards and opened below into pericard. No history, and no opportunity for physical examination.
54	Backward and to right	None	Reduced in capacity	Atheromatous	Atrophied. Bilobed aneurism.
55	General dilat. of inferior, and back-growth of superior aneurism	None	R. Dilated L. Dilated and hypertrophied	Atheromatous	Hypertrophied. Two aneurisms.
56	Forward and to left	None	..	Atheromat. in seat of aneurism	Aneurism extending forward and to left side, pressing on root of left lung, and causing absorption of ribs beneath left clavicle.
57	Superior forw. and inferior backward	Aortic slightly	..	Atheromatous	Two aneurisms.
58	Upward and to both sides	None	R. Dilated and hypertrophied L. Atrophied	Atheromatous	Left ventricle atrophied; the patient was in an advanced stage of phthisis.
59	General expansion	None	None	Highly atheromatous	No symptoms, and therefore no suspicion of aneurism; the man dropt down in the act of pumping water for the regiment.
60	Forward and to right	Slight thickening of aortic	R. Normal L. Concent. hypertrophy	Atheromatous	Not enlarged. Fatty accumulation. Severe and paroxysmal pain in right side of chest and arm, relieved by leeching.
61	Forward	None	Normal	Slightly atheromatous	Less than normal in size. Fatty accumulation. Aneurism, latent as to physical signs, but diagnosed from negative and rational evidence. Pneumonia, without physical signs.
62	Forward chiefly	None	Normal	Healthy, except at seat of aneurism	Atrophy. Severe paroxysmal pain in back and abdomen; large false aneurism
63	Downward and to right	None	Normal	Highly atheromatous	Not hypertrophied. Aneurism of ascending aorta opening into right ventricle. (See Case 69, p. 759.)
64	Backward, and to right	None	Normal	Atheromat. in seat of aneurism	Slightly enlarged (11 oss.)
65	Upward, downward, and laterally	None	R. Slight hypertrophy L. Slight hypertrophy and dilatation	Highly atheromatous	Slightly enlarged, and fatty accumulation. Large aneurism engaging entire arch, but latent.
66	Upward & backward	None	Normal	..	No hypertrophy. Fatty accumulation.
67	Upward & backward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied.
68	Forward, up. and backward	None	R. Dilated L. Hypertro.	Highly atheromatous	Hypertrophy of left ventricle, and fatty accumulation.

As illustrating the clinical history of thoracic aneurism, I beg to submit the following cases.

CASE CXXXV.—*Blow on Back ; Eighteen Months later, Pain in the Right Side of the Chest, followed by Pulsating Tumor beneath the Right Clavicle ; Double Impulse and Sound in Tumor ; Respiration Normal ; Murmur at Left Base ; Severe Pain in Scapular Region relieved by Leeching and Morphia ; Edema of Right Side, and Effusion into Right Pleura ; Sudden Death by Rupture of Sac and Extravasation into Right Pleural Cavity. Aneurism connected with Ascending Portion of the Arch of the Aorta, and containing Loose Clot ; Inferior Lobe of Right Lung Condensed by Effusion ; Slight Effusion into Pericardium ; Heart Fatty on Surface, but structurally Sound ; Aortic Valves slightly Thickened ; Left Ventricle in a state of Concentric Hypertrophy ; Aorta Atheromatous.*

William R., aged thirty-three years, of very intemperate habits, was received into hospital on the 12th day of February, 1866. Two years previously he had been knocked down by a cab whilst intoxicated. He received the blow upon the back, and was stunned by it, but he quickly recovered, and felt no further inconvenience from the accident till six weeks before admittance, when he was seized with pain in the upper part of the right side of chest; three weeks later he observed a pulsating swelling in the same situation. When he came under my notice, he was pale, but well nourished, and breathing freely and without difficulty in the recumbent posture. Pulse 84, regular, moderately full, and equal. Pupils normal and equal; no dysphagia. His only complaint was, of pain in the chest extending to the right shoulder. A prominence existed on the superior and anterior portion of the right side of the chest; it was semi-globular, closely resembling the female breast in figure, and about three inches in diameter at the base; it extended from a line half an inch beneath the clavicle, nearly to the level of the nipple, and from the right margin of the sternum to the middle of the clavicle. This swelling projected about an inch and a-half, was dull on percussion, and yielded a double shock; the first,

strong and coincident with the impulse of the heart, the second, diastolic in rhythm, and presented the character of a back-stroke during recoil of the tumor. The primary shock was stronger on the outer than on the inner side of the swelling, and was here distinctly excentric. No respiratory sound was audible over the projecting portion of the chest; but two sounds, remarkably clear, free from murmur, and synchronous with those of the heart, were heard. Over the remainder of the right side, and over the entire of the left side of the chest, percussion and respiratory sounds were normal.

The apex of the heart pulsated in the sixth intercostal space half an inch outside the nipple-line; precordial dulness was not extended. The first sound of the heart, as heard at the apex, was soft and prolonged; at the left base it was accompanied by a sharp "whiffing" murmur. This murmur was loudest in the area of the pulmonic orifice, but it was diffused universally over the left front of the chest; it could not be heard to the right of the mesial line, nor was it audible at the acme of inspiration or in the early period of expiration. Posteriorly, respiration was somewhat louder on the left than on the right side, and over both sides a double sound, synchronous with those of the heart and tumor, was heard. Neither impulse nor murmur could be detected posteriorly, nor was there anywhere along the spine a point of tenderness to pressure.

The diagnosis was not difficult. The sac of the aneurism was partially filled with fluid blood, for it was possible, by pressure, to reduce its volume considerably.

Whilst under treatment, the man complained occasionally of severe pain in the situation of the tumor, and extending to the right scapula. This pain, which was of a darting and intermittent character, deprived him for the time, of sleep; it was, however, always promptly and effectually controlled by the application of one or two leeches over the seat of its greatest intensity, followed by a draught containing ℥xl of the pharmacopœial solution of hydrochlorate of morphia. The pulse varied from 78 to 96, and was regular. Decubitus was dexter, and hence, there was slight œdema of the right shoulder and axilla. The carotid arteries pulsated visibly, and yielded a double sound but no murmur.

The abdominal aorta pulsed strongly, and readily yielded murmur on gentle pressure.

The man's condition underwent very little change between the 12th of February and the 14th of March, during which period he had been taking iron. On the last mentioned day, I remarked that the aneurism had advanced considerably in the direction forwards and outwards; it had become more solid, and its pulsation was stronger. The cedema of the right side now extended to the forearm, where the patient complained of sharp pain. Pulse 96. The iron was now stopped, digitalis was given, and two leeches were applied in the right axilla.

On the 19th, the pain was so severe as to induce copious perspiration; breathing was embarrassed even in the sitting posture, and the tumor had become more prominent.

On the 23rd the lower portion of the right side was dull, and respiration here was feeble and slightly bronchial.

At half-past six o'clock on the morning of the 25th, he called for his medicine, declaring he had had an unusually good night and felt much better; in the act of sitting up in bed he suddenly complained of agonizing pain in the abdomen, cried out that he was dying, was agitated, and became deadly pale; the tumor immediately ceased to pulsate, and the man died of syncope within thirty minutes.

The body was examined twenty-eight hours after death. The right side, measured over the prominence, exceeded the left in girth by three inches and a-half. The right pleural cavity was full of coagulated blood and serum; the clots alone weighed three pounds. On removing the blood, an enormous aneurism, as large as the head of a new-born infant, was seen to occupy the upper portion of the pleural cavity. It sprang from the upper part of the middle mediastinum, projected forwards and outwards, contracting adhesions to, and thrusting forward, the anterior wall of the chest, through which it had ultimately protruded by erosion of the second, third, and fourth ribs.

The aneurism was invested by the pleura in the greater portion of its circumference, and was unsupported, except at the root and inferiorly, where the middle lobe of the lung was attached to it. Below and behind the point of attachment just

mentioned there was a wide and irregular rent in the sac, through which two fingers might be passed; from this opening projected a large and jagged mass of partially decolorized clot. The superior and middle lobes of the lung were firmly attached to, and expanded upon the tumor, but they contained some air; the inferior lobe was entirely devoid of air, of a dark slate-colour, and pressed into the inferior and posterior portion of the pleural cavity.

The left lung was vascular on the surface, and partially emphysematous on the anterior margin, but otherwise healthy.

The pericardium contained about two ounces of serum; the heart presented a good deal of superficial fat at the base, and a thick layer in front of the ascending portion of the arch of the aorta.

On the right auricular appendix there was a large "milk-spot," and another existed in the usual situation on the right ventricle. A nodule of lymph, as large as a pea, was found attached to the left ventricle near the apex. The right chambers were normal; the left ventricle was much thickened, but diminished in capacity. The valves were all healthy, except those of the aorta, which were thickened and slightly incompetent.

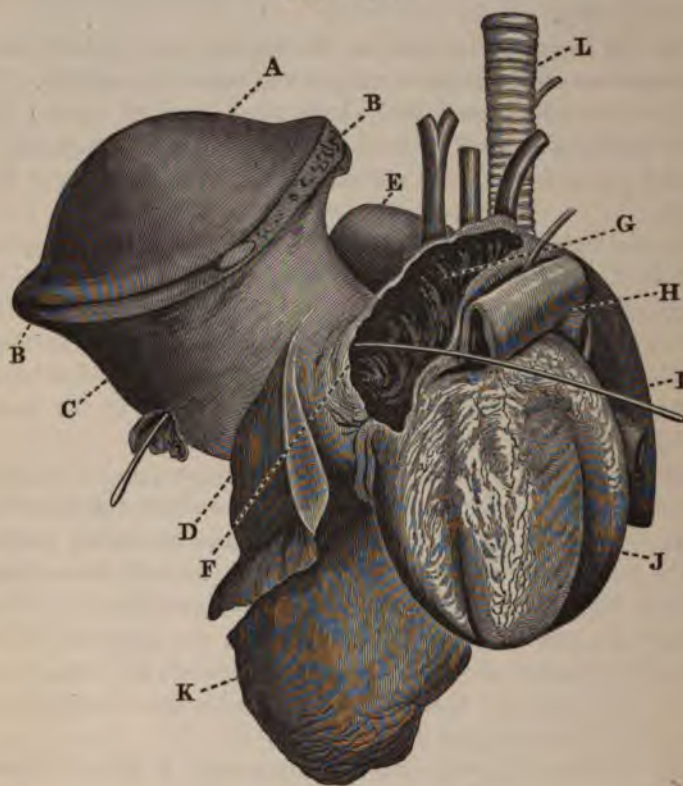
The lining membrane of the aorta was rugous, and of a dirty yellow colour; a circular opening, which admitted the points of three fingers, existed in the right wall of the ascending portion of the vessel, one inch below the origin of the arteria innominata, and led into the large aneurism previously described. The sac contained a good deal of coagulum, which was attached to the anterior wall, and projected through the rent on its inferior surface. This rent was distant about two inches from the orifice of communication with the aorta.

The pulmonary artery and its valves were healthy, as were likewise the primary branches of the aorta. The œsophagus, the descending cava and its tributaries, and the right bronchus, had escaped pressure; the pneumogastric nerves seemed much thickened.

The result of physical examination on the 23rd proved the existence, at that date, of serous effusion into the right pleura.

To this was added, two days later, the enormous volume of blood found in the pleura, by extravasation from the ruptured sac of the aneurism.* The subjoined woodcut (Fig. 69) from a coloured drawing by Mr. W. Laffan, a student of medicine, exhibits the aneurism and the adjacent parts in their relative position.

FIG. LXIX.



Aneurism of ascending portion of arch of aorta.

William R.

* Vide *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., p. 32, new series. I no longer admit, as I then stated, the existence of concentric hypertrophy of the left ventricle as a pathological entity, nor hypertrophy of that chamber as a consequence of aneurism of the aorta.

- A Aneurism projecting through chest-wall ; the integuments, fat, and pectoral muscles having been removed by dissection.
- BB Section of sternum and ribs encircling the projecting aneurism.
- C Intra-thoracic portion of aneurism, exhibiting a rent in its posterior inferior wall, by which the fatal hæmorrhage had occurred, and through which a jagged clot and the end of a bent probe are seen to project.
- D Middle lobe of right lung adherent to aneurism.
- E Apex of right lung projecting above and behind aneurism.
- F Aperture in aorta leading into aneurism. A bent probe is represented as passed through the openings of entrance and exit of the aneurism.
- G Aorta laid open by removal of its anterior wall, exhibiting the atheromatous condition of its coats, and the great vessels arising from it.
- H Left pulmonary artery and bronchus ; the pneumogastric nerve is seen coursing down behind them.
- I Descending portion of arch, and thoracic aorta, crossed by one of the left pulmonary veins.
- J The heart, exhibiting much fat on its anterior surface.
- K Inferior lobe of right lung compressed by the blood effused in the fatal hæmorrhage.
- L The trachea and left recurrent nerve.

The following measurements of the left ventricle were obtained ninety-six hours after death :

Thickness of Walls.

Near apex	$\frac{3}{4}$ inch.
Middle portion	$\frac{1}{2}$ "
Near base	$\frac{3}{4}$ "

Dimensions of Cavity.

Length from root of mitral valve to apex	...	2 inches.
Width at central portion, measured from septum	...	$\frac{1}{2}$ "
Width at base	do. do.	$\frac{3}{4}$ "

It thus appears that the left ventricle was in a state of concentric hypertrophy. The increased growth of the walls was due to functional compensation, rendered necessary by the atheromatous change of the aorta which had preceded the development of aneurism ; whilst the diminution of capacity depended upon spasm of the left ventricle, excited by the nervous shock arising from rupture of the aneurism, and favoured by the sudden withdrawal of vascular resistance in front.

CASE CXXXVI.—*Shooting Pains in Chest, ultimately fixed in Left Side; Defective Motion and Absence of Respiration on Left Side, and subsequently Pneumonic Solidification of the Left Lung without the usual Physical Signs; Slight and Temporary Dysphagia; Cough not Laryngeal; No Stridor; Temporary Improvement; Sudden Death by Rupture of Sac simultaneously into Left Bronchus and Œsophagus. Aneurism connected with Upper Portion of Descending Thoracic Aorta, and pressing upon Left Bronchus; Aorta Atheromatous; Effusion into Pericardium; Heart Fatty on Surface but less than average in size, its Walls and Valves Healthy; Left Lung in Third Stage of Pneumonia.*

John S., a hatter, aged thirty-four years, temperate, was received into hospital for me on the 19th of September, 1868. He stated that, nine months previously, he had been attacked with pain shooting across the front of his chest, and extending downwards on both sides; four months later, this pain became fixed in the left side. He had continued at his business till the date of his admission. He lay habitually on the left side, and had never spat blood.

State on admittance: Patient pale; no cough. Pulse 96, regular, and equal in both arms; pupils normal and equal. He complained of a dull aching pain on the left side, but shifting occasionally to the epigastrium. The right temporal artery was full and tortuous, and pulsated strongly; whilst the left temporal artery pulsated feebly, and was not visible. A corresponding difference was observable in regard to the pulsation of the carotid arteries on the two sides. Respiration tranquil.

Both sides of the chest were perfectly and equally clear on percussion; but motion was defective on the left side, and respiration was nowhere audible on that side, except at the sterno-clavicular articulation, and in the supra spinous fossa, in both of which situations an interrupted and harsh respiratory sound was heard. Respiration was puerile on the right side, and vocal vibration existed on both sides. No venous engorgement; no dysphonia, stridor, or dysphagia.

Treatment: Dry-cupping, and subsequently a few leeches at

seat of pain. Iron with chloric ether, and gr. j of aqueous extract of opium at night.

On the 22nd October the report was: Slept well and was free from pain last night; can now lie on either side; no change in percussion or respiratory phenomena. No impulse anywhere perceptible over the chest, except in the cardiac region, where it is normal. He was discharged on the 31st of October, nearly free from pain, and much improved in his general health.

I visited him at his lodgings on the 28th of November. During the preceding week he had been suffering from teasing cough attended with slight mucous expectoration, and had on several occasions vomited his food half an hour after taking it. He likewise felt obstruction in swallowing, above the pit of the stomach. The face was of a leaden tint. Right temporal artery full and tortuous; left not so. Pupils normal and equal. Pulse 108, weak but regular, and equal in the right and left arms. (See Figs. LXX. and LXXI.) The left infra-clavicular space,

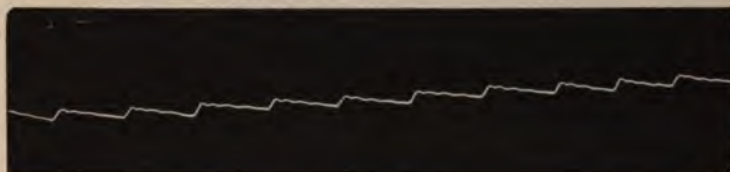


FIG. LXX.

Aneurism of descending aorta. Right radial pulse. John S.

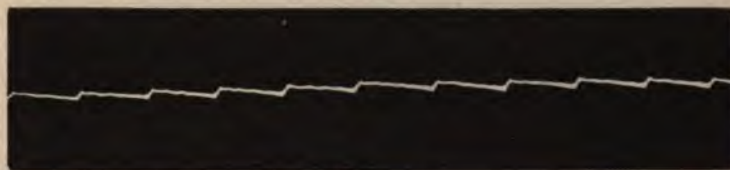


FIG. LXXI.

Aneurism of descending aorta. Left radial pulse. John S.

for a distance of two inches and a-half downwards, yielded the cracked-pot sound with muffled tympanitic resonance on percussion; below this level, as far as the nipple-line, there was comparative resonance, whilst over the remainder of the side

percussion-sound was normal. No respiratory sound was anywhere audible on the left side, except in the interscapular space, where it was feeble and bronchial. In the first intercostal space on the left side, close to the sternum, a distinct impulse was felt during forced expiration, over a space of one inch in diameter. To have gr. iss of the aqueous extract of opium at night.

He was readmitted on the 3rd of December. The apex-pulsation of the heart, which was very feeble, was then felt in the fifth intercostal space, whilst in the third and fourth interspaces, an inch and a-half inside the nipple-line, a strong pulsation, synchronous with that of the apex, was felt; the sounds of the heart were normal, but in the last mentioned situation the second sound was double. Both sounds were very faint in the course of the aorta. There was complete and uniform dulness of the left side as far as the left sterno-clavicular line. To the same extent vocal vibration was absent; respiratory sound was likewise in abeyance except in the left interscapular space, where it was bronchial, and at the left sterno-clavicular joint, where transmitted tracheal breathing was heard. Motion of left side imperfect. No abnormal impulse was anywhere perceptible. There was teasing cough, which was slightly laryngeal.

On the 15th, his condition had undergone improvement. The iron mixture and an anodyne at night had been continued. He walked about the corridor in the early part of that day, dined upon a chop at 1 o'clock, and swallowed without difficulty; he complained of no pain. At 9.30 o'clock, whilst in bed, he uttered a loud scream, and immediately afterwards vomited a large quantity of blood, and whilst in the act of reaching for a basin to the adjacent stand, he fell upon the floor; here, a few seconds afterwards, he was found in a state of syncope, but still conscious, and surrounded by a pool of florid and partially coagulated blood. He died fifteen minutes subsequently.

On examination of the body, the left lung was found to be solid throughout, except to a slight extent along the anterior edge, which was emphysematous and slightly congested; elsewhere the lung was hepatized, and of a marble tint; on section it was found to be infiltrated with pus. The right lung was voluminous and partially emphysematous; there was no appear-

ance of tubercle in either lung, and no pleural effusion. The pericardium contained about six ounces of serum. The heart was below the average size of the organ in the adult male, and was covered by a thick layer of fat. The chambers were all empty, and the valves were normal. The aorta was slightly atheromatous throughout the arch, and the left carotid was compressed at its origin by a large and hard lymphatic gland. Several glands similarly altered were found at the roots of the lungs.

Two inches below the origin of the left subclavian artery there was a large rent in the aorta, extending over three-fourths of its circumference, and leading into an aneurism as large as an orange. The interior of the sac was lined by laminæ of dense fibrin, half an inch thick, except anteriorly and to the right side where the mediastinal pleura alone formed its wall. Here a communication had been established between the aneurism and the left bronchus by an opening of the size of a pea; this opening presented jagged and sloughy edges, and was partially plugged by a shred of fibrin which was connected with the laminæ within the sac. On the right wall of the sac, and half an inch distant from the former opening, another, which was as large as a goose-quill, and quite patulous, existed. This latter opening led into the œsophagus, the mucous membrane of which was eroded to some distance around it. The appearance presented on the œsophageal aspect was that of a funnel, of which the small end was formed by the aperture leading from the bottom of the ulcer into the sac.

The diagnosis of aneurism of the descending portion of the arch, or of the upper portion of the descending thoracic aorta, pressing upon the left bronchus, was made at an early period of the case.

The temporary pressure upon the œsophagus is worthy of notice. It is probable that, for a week preceding the 3rd of December, when the man complained of obstruction in swallowing, accompanied by pain "above the pit of the stomach," the mucous membrane of the œsophagus had been undergoing erosion over the prominence caused by the ingrowth of the aneurism.

From the absence of hæmoptysis and hæmatemesis preceding the fatal hæmorrhage, it must be presumed that no communica-

tion between the sac and the gastro-pulmonary tract had existed during this period; whence it would follow, that the opening into the left bronchus, and that into the œsophagus, had been suddenly and simultaneously established a quarter of an hour before death. The existence of an effective plug in the opening which led into the bronchus, renders it more than probable that the fatal hæmorrhage occurred through the œsophagus.*

CASE CXXXVII.—*Patient Intemperate; Severe Strain of the Back followed by Dorsal Pain; Two Years later, Pain of a Paroxysmal Character in Epigastrium, Sides, and Back, and a Fixed Pain in Left Gluteal Region; Systolic Bellows-murmur at Epigastrium, and occasionally a Diastolic-murmur in the same situation, but no Impulse; Relief under Ordinary Treatment; Return of Severe Paroxysmal Pain in Left Side of Abdomen, and extending to the Left Hip and Knee; Tenderness and Murmur over Dorsal Spine; Excruciating Pain in Back which was relieved by hypodermic use of Morphia; Enlargement of Left Side; Absolute Dulness of its Lower Portion, with Hæmoptysis and Heaving Impulse; Upward Displacement of Heart, and Forward Displacement of Liver; Tympanitic Resonance of Apex of Left Lung; Partial Suppression of Urine; Paroxysmal Dyspnœa; Death by Exhaustion. Autopsy: Large False Aneurism arising from Inferior Portion of Thoracic Aorta, occupying nearly the entire Left Pleural Cavity, and extending Beneath the Diaphragm as far as the Left Kidney; Erosion of Three of the Dorsal Vertebrae.*

William F., aged twenty-eight years, a cabman of intemperate habits, first came under my notice about January, 1866, suffering from chronic laryngeal symptoms, manifestly the result of his irregular life. After a short course of treatment in hospital he was restored to his ordinary health, and discharged. Shortly afterwards he strained his back in lifting his cab, and then, for the first time, complained of pain in the lower dorsal spine. He

* Vide *Proceedings of the Pathological Society of Dublin*, new series, vol. iv., part i., p. 3.

became an extern patient of the Mater Misericordiæ Hospital on the 22nd of June, 1868, and was re-admitted soon afterwards. He then suffered from severe pain of a paroxysmal character in the epigastrium, and extending to the sides and back, and of fixed pain in the left gluteal region. Pulse 84, and weak, but regular, and equal on the two sides; pupils normal and equal; cardiac impulse strong; in the scrobiculus cordis a loud bellows-murmur was heard, which was synchronous with the pulsation of the abdominal aorta. Occasionally, a second murmur was likewise heard in this situation, diastolic in time, cooing in character, and resembling very closely a musical venous murmur. The former was faintly heard over the left costal border near the xyphoid cartilage, but evidently by transmission. The treatment consisted in iron given in a bitter infusion, aperients, and a small blister to the gluteal region.

Discharged, much relieved, July 31st, and again admitted August 14th. For two days after leaving hospital he had been entirely free from pain, but during the remainder of the period to the date of re-admittance he had suffered from excruciating pain in the left side of the abdomen, shooting down to the left hip and also to the left groin and knee, in the course of the ilio-scrotal and anterior crural nerves. This pain was paroxysmal, causing a "burning" sensation in the left groin and back, and was aggravated when he assumed the erect posture. A *bruit de soufflet* was heard beneath the ensiform cartilage, synchronous with the pulsation of the abdominal aorta; it was soft and lisping in character, traceable upwards to left of ensiform cartilage, where both sounds of the heart were likewise audible; here it was heard to follow the first sound, but at an appreciable interval of time, whilst it immediately preceded the second sound without any interval. After a voluntary movement of the patient's body the murmur was likewise audible at the apex of the heart, where it was fainter, but otherwise unaltered, and held the same relationship to the sounds. Its point of maximum intensity was at the tip of the ensiform cartilage; here it was constant, and when audible in all three situations, might be followed from below upwards and to the left in an uninterrupted line of about three inches, but with decreasing intensity from the ensiform

cartilage upwards. It was not audible elsewhere in front, and was independent of respiration. Above the level of the xyphoid cartilage the murmur was occasionally suppressed during one or two pulsations, and on two several occasions the most careful examination in the recumbent posture failed to detect it beyond the limits of the scrobiculus cordis. Occasionally, a second murmur was heard immediately succeeding the former, and therefore coincident with the systole of the aorta; it was musical in character, and strictly localized. Pressure upon the abdominal aorta at any point below the seat of murmur had the effect of diminishing the force of the principal, and of suppressing the second or after-murmur. The heart pulsated in its usual situation, and the area of precordial dulness, and the cardiac sounds, were normal.

To have two leeches to seat of pain in epigastrium.

August 23rd. No murmur audible anywhere in front, save in the scrobiculus cordis, where it is double; the latter element occasionally musical.

24th. The principal murmur is again audible over line from apex of heart to scrobiculus. Over the last dorsal and first lumbar spinous processes, and to an extent of two inches around this point, a loud blowing murmur, but unattended with impulse, is heard. This part is tender on pressure, and the seat of "shooting," "burning" pain. To have two leeches applied here.

On the 2nd of September the pain on the left side of the abdomen and back was excruciating; the patient implored us to relieve him, and one-twelfth of a grain of muriate of morphia in solution was injected subcutaneously in the former situation, with the effect of completely subduing pain for a period of twelve hours.

The following remarks, written under date of 3rd September, are copied from my note-book:

"The collateral evidence here is sufficiently strong to warrant the diagnosis of inferior thoracic, or thoracico-abdominal aneurism, even in the absence of tumor and perceptible impulse. No doubt exists in my mind as to the non-cardiac nature of the murmur heard over the left costal cartilages, and as to its iden-

tity with that heard beneath the ensiform cartilage and synchronous with the pulsation of the abdominal aorta, though modified by distance from the seat of its origin.

"The interval of time that must elapse between the systole of the left ventricle, and the manifestation of its effects, by impulse and murmur, in an aneurism of the inferior thoracic or superior abdominal aorta, though 'infinitesimal, is nevertheless strictly calculable from a knowledge of the distance between the points mentioned, and of the rate at which the blood-wave moves in the individual under examination.* If the interval so determined be appended to the period of the first sound of the heart, and the abdominal murmur which marks it be supposed to be transferred or transmitted to the area within which the cardiac sounds are audible, it is manifest that the murmur will be heard in the precordium at the close of that interval, and that it must coincide with some one cardiac phenomenon posterior in time to the first sound.

"Now, if that phenomenon happen to be the negative one of the short pause, and the terminal portion of it, we shall have a murmur, not belonging to the heart, audible in the precordium, and coinciding with that portion of the short pause immediately anterior to the second sound. Such a murmur I would designate as *prediastolic*.

"In the case under consideration, if one hand of the observer be placed upon the abdominal aorta, and the other in the site of the apex-pulsation, whilst the ear is applied to the stethoscope placed below the ensiform cartilage, the asynchronism between the impulse of the heart upon the one hand, and the pulsation of the aorta and the murmur on the other, will be readily perceived."

Murmurs of this precise rhythm, not located at the aortic orifice, are invariably exo-cardial.

The patient's condition underwent some improvement, and he was discharged in the early part of October. I now lost sight of

* Dr. Handsell Griffiths (*British and Foreign Medico-Chirurgical Review*, October 1868) has, as the mean of the results obtained from 300 experiments with his hæmadromometer, estimated the velocity of the blood-wave in the carotids of the dog at 400 millimètres (16 English inches) per second.

him till March, 1869, when I visited him at his lodgings. He was then coughing up unmixed florid blood in considerable quantity. Pulse 120, regular, and equal; pain was now referred chiefly to lower part of left side of chest, which was absolutely dull, and devoid of vocal thrill and respiratory sound; the intercostal spaces were here obliterated, but tender to pressure, and the hand placed on this portion of the chest perceived a general heaving movement.

From the angle of the scapula upwards respiration was normal but accompanied with râles; a faint, rough systolic* murmur was audible at the ensiform cartilage; the heart pulsated above the level of the nipple, and inside the nipple-line; its action was strong, and its sounds loud, clear, and normal.

A blister was directed for the side, and acetate of lead in gr. v doses as a styptic. On the 18th of March he was again admitted into hospital, and on the 19th, the following note of his condition was taken: Pulse 120; left side absolutely dull at all points below level of second intercostal space, and devoid of respiratory and vocal phenomena; impulse faintly perceptible here; above level of third rib percussion-resonance is of a hollow tympanitic character; in front, this part of the chest yields no respiratory sound, but posteriorly it is the seat of bronchial respiration with fine crepitus; over posterior inferior portion of left side two sounds are heard, but no murmur; and at the scrobiculus cordis a murmur is audible only on making firm pressure with the stethoscope. The patient was sleepless, and expectorating viscid pneumonic sputa; the lips were dotted with the crusts of dried herpetic eruption. To have gr. iss of aqueous extract of opium at night.

On the 9th of April it was noted that the left infra-clavicular region, from the median to the axillary line, had continued to yield a hollow tympanitic sound. Respiration had been for some days remarkably variable; at one time it was loud and puerile, and accompanied by coarse muco-crepitus, and on the following day all but suppressed; these alternations were of frequent occurrence. The heart was now displaced upwards, and pulsated visibly and

* *I.e.*, representing the *systole* of the left ventricle in a *distant* artery, in the sense previously explained.

tumultuously in the second and third left intercostal spaces, near the margin of the sternum; these spaces, nevertheless, yielded a hollow sound on percussion. The entire left side was enlarged, and the inferior costal cartilages of that side projected considerably; there was likewise a prominence in the inferior axillary region, and the patient was troubled with a loud ringing cough. The upper portion of the sternum, with the corresponding extremities of both clavicles, projected sharply, but this projection had existed from childhood, and was due to the natural conformation of his chest. There was no unusual dulness in this situation, but at the acme of expiration an obscure heaving pulsation was perceived by the hand placed flat upon it; and over its lower and right portion, corresponding to the attachment of the second costal cartilage to the sternum, and strictly limited to a space of about two inches in diameter, a harsh murmur, prediastolic in rhythm, was heard. The cardiac sounds were loud and free from murmur. The epigastric hollow was obliterated; the liver projected considerably, but equally; but its surface was even; the organ was not displaced downwards, nor did the hepatic dulness extend above the level of the sixth rib. Expectoration copious and purulent, and occasionally mixed with a little blood.

29th. The secretion of urine has been greatly diminished since last report, amounting for the last fortnight to not more than eight ounces in the twenty-four hours; it is loaded with lithates. Pulse 120, weak, but regular; respiration 48; cough troublesome and ineffective; loud puerile respiration for two inches below the left clavicle. A faint murmur, synchronous with abdominal pulsation, existed at the ensiform cartilage, where the sounds of the heart are faintly audible, but from this point upwards to the site of cardiac pulsation they increase progressively in distinctness. On the preceding night, the patient had been attacked with aggravated dyspnoea, accompanied with severe pain in the epigastrium, shooting down to the left groin. To have a sedative of hydrocyanic acid and morphia at night.

On the 6th of May a loud bellows-murmur, nearly coincident with the radial pulse, was detected in the sitting posture along the lumbar spine, and for two inches to the left; in this situa-

tion there was severe and fixed pain, which was not aggravated by pressure or by percussion; prediastolic murmur, remarkably loud and harsh over sternal prominence, and somewhat to right, in both of which situations the hand can detect a heaving impulse; between the site of this murmur and that of cardiac impulse no abnormal sound was discoverable; neither did murmur exist in connexion with the cardiac sounds. The pulsation of the heart was violent, lifting up at each stroke the corresponding portion of the chest-wall, which yielded a hollow sound on percussion. No respiratory sound was audible in left infra-clavicular space; there was some fetor of the breath. To have compound decoction of scopolarium with nitrous spirit of ether; also an anodyne at night.

9th. During my visit to the ward at 12 o'clock he was suddenly attacked with the most distressing dyspnoea, which caused him to start into the sitting posture; respiration was very rapid and laboured, and the cervical veins became turgid, but the face remained pale. On inquiry I learned that in the course of the preceding fortnight he had had several such fits, but none of so aggravated a character as this; there was great anxiety, and the patient besought me not to leave him. The dyspnoea continued, unmitigated by ethers and stimulants; the patient became gradually weaker, and died of exhaustion at 2 o'clock. On the evening of that day a *post mortem* examination of the body was made by the resident pupils, Messrs. Furlong and Kelly, under circumstances of much difficulty. To these gentlemen, and to Mr. Petit, I am indebted for the opportunity of exhibiting the morbid specimen to the Society,* and of clearing up some doubts that arose in my mind in the course of the case.

The haste with which the examination was made will account for some injuries sustained by the viscera during removal.

On removing the sternum a good deal of serum escaped; whence this proceeded it is difficult now to say, but it probably came from the right pleura. The great veins at the root of the neck, and the cavæ, were distended with blood; the heart lay across the chest below the left clavicle, its apex to the left and

* A report of this case was laid before the Medical Society, Wednesday, May 26th, 1869.

its base to the right side; it occupied the great pulmonary fissure, displacing upwards, and pressing into the cone of the pleura, the superior lobe of the left lung, the base of which overlapped and in great measure concealed it from view; it was rather below the average size of the adult heart, and was perfectly sound in its walls and in its valves. The arch of the aorta was free from disease, but the curve at the junction of its ascending and transverse portions was greatly exaggerated, owing to the displacement of the heart upwards. The superior lobe of the left lung was universally and firmly attached to the parietal pleura, and much reduced in volume by compression between the displaced heart and the superior osseous boundaries of the chest, but it was otherwise free from disease. The inferior lobe of the lung was entirely obliterated as such; it was converted into a thin lamina spread out upon, and firmly attached to, the surface of a vast, solid tumor which occupied nearly the entire of the left side of the chest. This tumor was found to be an aneurism, communicating with the thoracic aorta immediately above the diaphragm by an oblong opening about two inches by one inch in diameter, and engaging one-half the circumference of the vessel; the edges of this opening were rounded off, and expanded into a vast aneurismal pouch which extended vertically from the second rib to the left kidney, displacing the lung and heart upwards, the diaphragm downwards, the liver forwards, and the ribs and costal cartilages outwards. It was firmly attached to the chest-wall, from which it was removed with great difficulty, and inseparably so to the diaphragm beneath the external arched ligament; thence it had descended behind the left kidney, and was attached to the upper extremity of that organ. The sac of the aneurism, which was formed by condensation of the surrounding tissues, was lined on three aspects by dense laminated fibrin; viz., inferiorly, externally, and posteriorly; but superiorly, and also internally, it was composed of little more than the pleura; whilst anteriorly, as already stated, it was strengthened by the condensed and adherent lung-tissue. The sac contained a great quantity of dark clotted blood, and in its upper part, a mass of shreddy fibrin mixed with coagulum.

There was no opening from the sac save that which communicated with the aorta.

The bodies of the sixth, seventh, and eighth dorsal vertebræ were deeply and extensively eroded ; the seventh had been almost entirely destroyed. The corresponding intervertebral fibro-cartilages were scarcely affected. The œsophagus, trachea, and bronchi had escaped pressure. The right lung was healthy, as were likewise the liver, spleen, and kidneys ; but the left renal artery was pressed on and nearly obliterated by the tumor.*

In cases of this kind little can be done curatively ; indeed nothing beyond mitigating the sufferings of the unhappy patient and postponing the final event, whether from expansion or rupture of the sac, by means of regimen and appropriate medicine.† The interest connected with such cases, therefore, has reference mainly to diagnosis.

A short *résumé* of this case may suggest profitable reflection.

A young man of intemperate habits strained himself in lifting a weight ; shortly afterwards he complained of pain in the back ; two years subsequently he presented himself, and was then suffering from acute pain in the lower part of the back, which extended to the left side of the chest, the left hip, and groin. This pain was intermittent and paroxysmal, and described as being of a "burning," "boring," character, and aggravated by the patient's assuming the erect posture. A murmur was heard at the ensiform cartilage, posterior in time to the first sound of the heart, anterior to the second sound, and synchronous with the pulsation of the abdominal aorta ; a second murmur of a musical character, and coinciding with the systole of the aorta, was also heard occasionally in this situation ; the former of these murmurs was modified, and the latter extinguished, by pressure upon the abdominal

* *Vide* Fig. LXXII.

† Since the publication of this paper in *The Dublin Quarterly Journal* for August, 1869, I have been favoured with a copy of a very interesting and valuable pamphlet on the treatment of aneurism by means of iodide of potassium in large doses, by Dr. George W. Balfour, of Edinburgh. Since receiving the pamphlet, for which I beg to express my obligations to Dr. Balfour, I have made partial trial of his treatment, and feel bound, so far, to report favourably of it. In the only case of the kind in which I have since had an opportunity of administering the iodide, namely, one of abdominal aneurism, the excruciating pain promptly ceased after its exhibition.

aorta below the seat of their origin ; a single bellows-murmur was audible over, and in the vicinity of, the lower dorsal vertebrae, which were the seat of pain and tenderness, and the centre whence pain radiated into the chest and abdomen ; no impulse was discoverable in the epigastrium, and no murmur below the ensiform cartilage. The action and sounds of the heart were normal ; there was no febrile excitement ; vital functions performed satisfactorily, and general nutrition unimpaired. Five months subsequently, the left side of the chest was found enlarged, and dull on percussion, from the base to the angle of the scapula ; to the same extent respiration and vocal vibration were abolished ; the heart pulsated above and inside the left nipple ; respiratory sound was heard superiorly, and here percussion sound was hollow and tympanitic.

The patient was pale, expectorating pure florid blood, but free from hectic symptoms ; his pulse was quick and feeble.

Three weeks later, the liver was found pushed forwards into the epigastrium, but not increased in volume ; the heart was displaced upwards beneath the clavicle, the ribs were everted, and the inferior costal spaces obliterated ; a heaving pulsation, accompanied with a double sound, was at the same time perceptible over the inferior posterior portion of the corresponding side. The murmur at the ensiform cartilage persisted, but was then much less distinct ; a bellows-murmur, slightly posterior in time to the radial pulse, was heard along the lumbar spine ; renal secretion was partially suppressed ; the patient became gradually weaker ; paroxysmal dyspnoea supervened, and in one of these fits he expired.

The foregoing sketch, viewed in its entirety, no doubt leads necessarily to the diagnosis of aneurism of the inferior thoracic aorta. But it is no less manifest that certain portions of it, not interpreted by the light of the remainder, might seem to warrant a different conclusion. Thus, for example, if the patient were seen by a medical man for the first time when the left side of the chest was enlarged, dull on percussion, and devoid of respiratory and vocal phenomena throughout the greatest part of its extent, whilst superiorly there was persistent modified resonance with variable respiratory sound, the latter being, as repeatedly hap-

FIG. LXXII.



- 1-2 Superior lobe of left lung overlapping and attached to the heart. (1, Lung tissue in section.)
- 3 Heart (pericardium removed) occupying pulmonary fissure, and flattened by pressure.
- 4 Inferior lobe of lung flattened out and incorporated with aneurismal sac (seen in section.)
- 5 Aneurismal sac, torn in process of removal.
- 6 Jagged and coloured fibrin occupying aneurism.
- 7 External surface of sac formed by thickened pleura.
- 8 Empty portion of sac.
- 9 Diaphragm expanded upon and attached to sac.
- 10 Opening in aorta (two probes introduced into the vessel).
- 11 Everted portions of torn sac.
- 12 Left kidney.

pened, loud, and accompanied with coarse muco-crepitus, amounting almost to *gargouillement*, one day, and on the next entirely suppressed; the patient expectorating fetid, blood-stained pus, and the liver and heart displaced, the diagnosis of primary disease of the lung, probably malignant, with copious secondary liquid effusion into the pleura, would, not improbably, have been made.

The annexed engraving (Fig LXXII.) exhibits the aneurism in the foregoing case, and also the relative position and general appearance of the parts related to it.

CASE CXXXVIII.—*History of Intemperance and Injury of Chest by Strain; Cough, Orthopnœa, Stridor, and Paroxysmal Dyspnoea from Spasm of the Glottis; Imperfect Respiratory Movement on both Sides of the Chest, with almost complete Suppression of Respiratory Sound on the Left Side; Absence of most of the Collateral Symptoms, and Total Absence of the Proper Signs of Aneurism; Laryngotomy, followed by Complete Relief from all the Urgent Symptoms; Death more than a Month subsequently from Pneumonia. Autopsy: Aneurism arising from the Posterior Wall of the Transverse Portion of the Arch of the Aorta at its Left Extremity, and Pressing upon the Trachea; Necrosis of the Larynx, and Double Pneumonia.*

Thomas B., aged thirty-three years, by occupation a packer in the egg market, and very intemperate, entered the Mater Misericordiæ Hospital on the 13th of March, 1871. Three weeks previously, whilst carrying a heavy hamper of eggs, he was suddenly seized with pain in his back, and difficulty of breathing. A week later he entirely lost his breath after ascending a flight of stairs, but felt no pain, and at the end of another week a loud, wheezing noise accompanied his breathing.

Latterly, both the respiratory distress and the stridor had become more troublesome, and for the three weeks immediately preceding admittance he had had cough. He had never spat blood.

When admitted, he was pale, with a remarkably anxious ex-

pression of face; respiration was embarrassed; the patient was unable to lie down, and a faint stridor accompanied inspiration. There was cough, with expectoration of mucus, which, as he declared, frequently "gathered at the root of his neck," causing increased distress of breathing, and was brought up with much difficulty by coughing.

No venous engorgement or dysphagia. Pulse 84, small, regular, and equal on the opposite sides. Pupils normal and equal. Voice somewhat husky. Precordial dulness, site of apex-pulsation, and sounds of heart normal, but impulse feeble. No abnormal impulse, and no murmur anywhere discoverable. Respiratory movements were imperfect on both sides of the chest, but in a greater degree on the left side than on the right; respiration was likewise feeble, and masked by loud stridor on both sides. The slightest movement of the patient's body, such as that required in drawing on his clothes, was capable of inducing a paroxysm of the most urgent dyspnoea, accompanied by loud stridor. On examination with the laryngoscope, which was made with much difficulty owing to the distress which it occasioned the patient, the vocal chords were seen to move equally, and no tumor, œdema, or other cause of obstruction was visible.

The paroxysms of dyspnoea became gradually more severe and more frequent; they likewise now occurred irrespectively of effort on the part of the patient, but were in some degree alleviated by large doses (grs. xx) of bromide of ammonium.

On the 10th of April he had several paroxysms accompanied by loud stridor, each lasting about an hour, during which he partially lost consciousness. He experienced some relief from ℥xxx of wine of ipecacuanha, with ℥x of tinct. of cannabis Indica, given during the paroxysms.

On the 13th of April, the patient being nearly asphyxiated in a paroxysm of unwonted severity and duration, laryngotomy was proposed, but the poor man requested it should be postponed till the following day.

On the 14th, however, urgent dyspnoea with loud and shrill stridor having continued with unremitting intensity through the preceding twenty-four hours, laryngotomy was performed by my colleagues, Messrs. Cruise and Hayes, with much difficulty, owing

to the extreme restlessness of the patient. The cricoid cartilage and the first ring of the trachea were divided. Considerable relief followed the operation, as the patient lay down immediately afterwards and obtained some tranquil sleep; he likewise took some wine and water, which previously he had been unable to swallow, owing to the dyspnoea which the attempt occasioned.

16th. He had passed a good night, having slept several hours after a draught containing grs. xxv of chloral hydrate; respiration tranquil, and when the tube was closed with the finger, he could breathe freely through the glottis; he was likewise able to swallow liquids without difficulty. Pulse full and regular; slight bronchial râle; respiratory sound was loud and harsh on the right side, and almost absent on the left.

17th. Took chloral draught, and slept through the night after 10 o'clock; feels much better, and desires to know when the tube might be removed. After this date, tumefaction and ulceration appeared about the wound, and were much benefited by warm stuping, and dressing with oxide of zinc ointment, the edges of the wound having been protected from the pressure of the tube by means of strapping with adhesive plaster.

On the 29th he could breathe freely through the larynx, and speak in an audible voice; but he could not expectorate save through the tube. The expectoration, which had been previously copious and muco-purulent, was then much reduced in quantity. He could swallow solids with facility, and sleep in the recumbent posture. The bowels and kidneys were acting naturally. Respiratory murmur, which for two days after the operation had been all but inaudible in the left lung, was now equally distinct on both sides; the action and sounds of the heart were normal, the radial pulses were regular and equal, no abnormal dulness or pulsation was anywhere perceptible, and no pain or tenderness was referred to any point of the chest.

Shortly after the last mentioned date, the patient began to expectorate pus in large quantity. About the middle of June the sputa became pneumonic; pain was felt over the base of the right lung, and here the physical signs of pleuro-pneumonia were detected. Despite treatment, and with intervals of slight

improvement, the man's condition gradually became worse; he died, exhausted, on the 23rd of June.

On examination of the body, serum, to the amount of eight ounces, was found in the pericardium, but no evidence of pericarditis existed. A large "milk-spot," raised above the surface, and covered with epithelium, was found on the anterior surface of the right ventricle; another, of smaller size, existed on the right side of the apex. The heart was healthy in structure but slightly enlarged; it weighed eleven ounces. The valves were all in a normal condition. There was a good deal of decolorized fibrin in the right auricle, and in both ventricles, whence it extended into the aorta and pulmonary artery. The lower lobe of the right lung was covered with false membrane, and in the third stage of pneumonia; the inferior lobe of the left lung was congested and solidified.

The lining membrane of the aorta, throughout the arch and the descending portion, was of a deep crimson tint, and the left extremity of the transverse portion of the arch was dilated, and mottled with specks of atheroma. In the posterior wall of the vessel, directly behind the left subclavian artery, and about three lines from the orifice of that vessel, a circular aperture, three-fourths of an inch in diameter, existed, leading into an aneurism of the size of a bantam's egg. The edges of this aperture were even, but fissured by scaling of the lining membrane.

The aneurism projected into the antero-left wall of the trachea, which was here reduced to one-half its normal diameter, the wall of the trachea being much attenuated, and its lining membrane congested, at the point pressed upon. The sac was nearly empty, a few flakes of fibrin only adhering to its posterior wall; it was flaccid, and rugose on the internal surface. The left recurrent laryngeal nerve took its normal course, and both it and the œsophagus had entirely escaped pressure.

The cricoid cartilage was completely and universally necrosed; it fell to pieces, as small, dark, and gritty particles, during removal. The lower edge of the thyroid cartilage was likewise in a state of necrosis.

In this case, aneurism had been diagnosed from the occurrence of paroxysms of laryngeal dyspnoea, unaccompanied by disease of

the larynx, and preceded by a definite history of strain and injury within the chest. The precise seat of the disease had been likewise approximately determined, mainly from the feebleness of respiratory sound in the left lung. The principal interest connected with the case, however, arises from the relief obtained by laryngotomy.

CASE CXXXIX.—*Severe Neuralgic Pains, with Diaphragmatic Pleurisy ; Harsh Dry Cough ; no Special Symptom of Aneurism, and no Sign, except Sharp Second Sound in the Ascending Portion of the Arch of the Aorta ; Sudden Death. Autopsy : Hæmorrhage into the Pericardium by Rupture of a Large Dissecting Aneurism of the Aorta, engaging the entire of the Ascending and Transverse Portions of the Arch ; Fatty Accumulation upon the Heart, and almost Total Absorption of its Muscular Substance ; Valves not Affected.*

Mr. John B., aged sixty-two years, a man of great ability, and an architect by profession, had been, for more than three years, suffering from wandering pains, apparently rheumatic, in the muscles of the chest, back, and shoulders. On one occasion these pains were localized in the diaphragm, and associated with the signs of diaphragmatic pleurisy on the right side. During the whole of this period he had been under my care ; and a few months before his death, whilst suffering from pains referable to the diaphragm, he consulted Dr. Stokes. Neither of us detected any evidence of aneurism. Having repeatedly and carefully examined his chest, I can positively affirm that, with the exception of the neuralgic pains just mentioned, no symptoms suggestive of aneurism existed. Mr. B. was a man of very active habits, exceedingly nervous, and disposed to exaggerate his symptoms. He recovered from his last attack, that of diaphragmatic pleurisy, after a protracted illness, and resumed his professional duties ; but, as he informed me, he was no longer equal to his wonted activity, and fatigue of any kind told sensibly upon him. He continued, however, at his professional avocations, and was able to travel to distant parts of the country. A year later he called on me again, and complained of neuralgia

of the face and arms, but there was nothing in his condition to attract special notice. I treated him for neuralgia, and he seemed to have recovered his usual health.

He visited me, for the last time, on the 10th of November, 1871, to seek relief from neuralgia of the most aggravated character. He described the pains as shooting over the face, scalp, and neck, down the right arm, and around the right side of the chest; he pointed to one spot, midway between the right nipple and the sternum, as the seat of fixed pain. The pulse was 84, rather weak, but regular and equal; appetite good, and all the functions regularly and effectively performed. The pain usually ceased at night, and his sleep was unbroken and refreshing. The pupils were normal; he was not subject to vertigo or faintings, and had never spat blood; there was no arcus senilis, or œdema. He informed me that he had a harsh dry cough, which, however, I had not the advantage of hearing.

I made an examination of his chest, directing my attention only to the precordium and the seat of alleged pain to the right of the sternum. The action of the heart was quite regular, but the first sound was faint, whilst the second sound was remarkably sharp, loud, and abrupt at the base of the heart, and, in a still greater degree, at the right margin of the sternum, on a level with the second and third interspaces.

I made light of his case, and was about to prescribe for the neuralgia, which was the only symptom that seemed to demand attention. He was a very intellectual man, and both of us being interested in the subject of a passing conversation of the previous moment, I asked him a question in reference to it, whilst proceeding to write a prescription. He answered rather excitedly, as was his habit; I heard a rustling movement, and, turning round, I saw him fall off the chair towards the left side, and upon his face. It immediately occurred to me that he had been subject to epileptic seizures which he had hitherto concealed. I lifted him up, and at once saw that the matter was more serious. His neck became turgid, the pulse ceased, and he made a few gasping respiratory efforts. I turned him over on his back, having hastily summoned the servant from the adjoining hall; a dark shadow passed slowly over his face from below, manifestly

the result of reflux venous engorgement from the right side of the heart; and life had ceased. The whole period occupied by the occurrence just described did not exceed three minutes.

At the instance of the City Coroner, Surgeon N. C. Whyte, and with the assistance of my friend, Dr. E. W. Collins, I made a careful examination of the chest two days after death. The anterior mediastinum was occupied by large masses of yellow adeps; the pericardium was distended with serum and blood-clot, to the amount of a pound and a-half. The heart was somewhat enlarged, covered with fat, and remarkably soft and flabby. The aorta was greatly dilated immediately above the orifice, and the internal and middle coats, the latter of which was highly atheromatous, had given way, the blood insinuating itself between the middle and the external coat. The aorta had gradually expanded, from the sinuses of Valsalva upwards, into an aneurism of the size and shape of a pine-apple, capable of containing at least twelve ounces of blood, and engaging the entire circumference of the vessel as far as the descending portion of the arch. On the right side of this enlargement, half an inch above the valves, a rent was seen by which the blood found in the pericardium had escaped. This led upwards into a sinuous passage between the external and middle coats of the vessel, which extended over a considerable portion of its circumference, and ended in an irregular opening into the aorta nearly on a level with the reflection of the pericardium.

The internal surface of the sac, which contained some loose blood-clots, was rugose, highly atheromatous, and fissured in several places. The three primary branches of the aorta were strictly normal in diameter, and free, as was likewise that vessel itself, beyond the transverse portion of the arch. The trachea and the œsophagus had entirely escaped pressure. The left ventricle was dilated and hypertrophied, the outer two-thirds of the substance of its walls consisting of fat, which had accumulated on the surface of the heart and grown inwards, displacing and causing atrophy of the muscular substance.*

The internal portion of the ventricular wall, seen in section, presented the normal appearance to the naked eye; its fibres

* *Vide* Fig. XXXIV., p. 608.

exhibited under the microscope the ordinary transverse striæ.* The fat cells were large, and partially collapsed, through the action of sulphuric ether in which the section examined had been previously soaked.†

The right ventricle was in a more advanced stage of fatty substitution; the entire thickness of its wall, except a film on the internal surface not thicker than a threepenny piece, having been replaced by fat. The right auricle was laden with fat, very

FIG. LXXIII.



Aneurism of arch of aorta and fatty heart. Mr. John B.

* *Vide* Fig. XXXVI., p. 609.

† *Vide* Fig. XXXV., p. 609.

little of its muscular substance remaining. The valves were all healthy and competent. The most noteworthy feature in this case is the latency of a twofold disease of so formidable a character as aneurism of the aorta with fatty transformation of the heart. With the exception of harsh and dry cough, which I had not heard, and migratory neuralgic pains, localized, however, at one point of the chest, there was absolutely no symptom suggestive of aneurism, and none which would have directed special attention to the thorax as the seat of organic disease of any kind. The dull character of the first sound, and the sharp ringing quality of the second sound, excited a passing suspicion of weak heart and atheroma of the aorta; but, in the absence of more definite evidence, neither aneurism nor fatty disease of the heart was contemplated. Complete and careful examination of the chest was, therefore, not made on the occasion of the patient's last visit to me.

The annexed engraving (Fig. LXXIII.), from a drawing by Mr. Burnside, shows the appearance presented by the heart and the aneurism after dissection. The sac had been laid open; the uneven condition of its internal surface is represented. The openings of the three primary branches of the arch are indicated by bristles, and the continuation of the aorta, by the passage of a piece of gum elastic bougie.

CASE CXL.—*Aphonia, Stridor, Orthopnoea, and Paroxysmal Dyspnoea; Suppression of Left Radial Pulse, and Contraction of Left Pupil, with Ptosis; Subclavicular Dulness, and Feeble Respiration on Left Side; Copious Expectoration of Pus; Death by Asphyxia. Autopsy: Emphysema of both Lungs, and Pneumonia of Right Lung; Deposit of Fat upon Heart; Aneurism of Transverse portion of Arch of Aorta pressing upon Left Subclavian, displacing and stretching the Oesophagus, Right Pneumogastric and Left Recurrent Nerves; Larynx Healthy.*

Mrs. L. I., aged thirty-one years, entered the Mater Misericordiae Hospital on the 2nd of January, 1865. She had enjoyed

good health till the early part of the preceding month, when, after exposure to cold, she was attacked with cough, and entirely lost her voice. For these symptoms she had been under treatment in one of the city dispensaries, and, deriving very little benefit therefrom, she sought relief in hospital.

Her appearance indicated great suffering; she was thin and haggard, and her face was pallid, with a tinge of lividity. There was complete aphonia, with orthopnoea, and loud stridulous breathing, audible at a considerable distance from the patient. The slightest movement of her body, and even the exposure of her chest for the purpose of examination, was immediately followed by a paroxysm of coughing, accompanied by copious expectoration of frothy mucus. The right radial pulse was 108, and weak, but regular; whilst in the left brachial and radial arteries no pulsation could be detected. With a suspicion of intra-thoracic tumor before my mind, I made a careful examination of the chest, which, owing to the intensity and general diffusion of bronchial stridor, I found it impossible to carry out to my entire satisfaction. The only signs of a positive kind which could be detected were slight dulness under both clavicles, and comparatively feeble respiration in the left infra-clavicular space. On the day subsequent to that of admission, I tested the power of swallowing, and found it to be unaffected.

On the 7th, I observed a marked contraction of the left pupil, which continued in the shade. The larynx, examined with the laryngoscope, was found to be structurally healthy, and the vocal chords were seen to move normally in respiration. At my request, Dr. Hughes made a careful examination of the patient; he declared that, except contraction of the pupil, and suppression of the radial pulse (both on the left side), he could discover no evidence of intra-thoracic tumor. Dr. Hughes detected feeble pulsation in the left brachial artery.

On the 8th, partial ptosis likewise existed on the left side, the upper lid drooping to the level of the equator oculi. During the fits of spasmodic dyspnoea and cough which were consequent upon the slightest movement or exposure of the patient, pus was expectorated in large quantity, to the momentary relief of the patient.

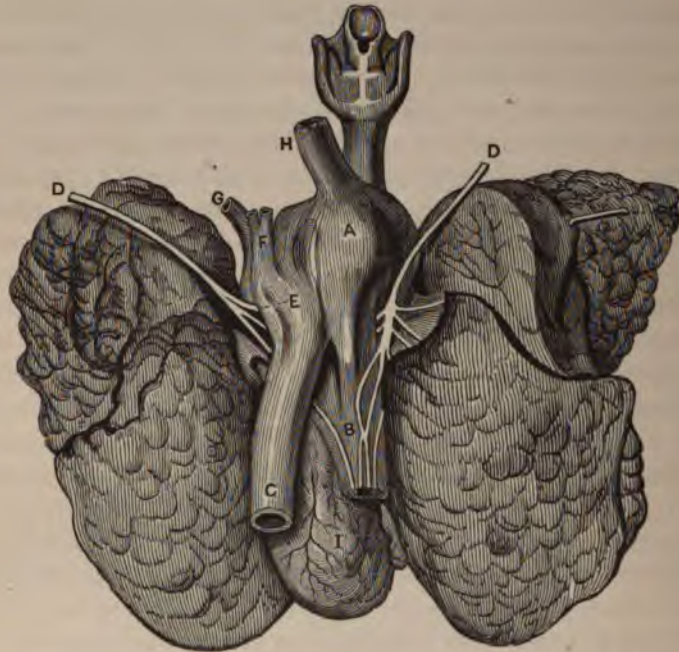
The pulse became weaker, the respiration more rapid, and, notwithstanding that food was taken in moderate quantity, the patient sank very quickly. She died on the night of the 15th in a paroxysm of dyspnœa.

On examination of the body, both lungs were found to be emphysematous; the left being attached to the chest-wall by recently effused lymph and its base in the third stage of pneumonia. The larynx was healthy. There was a deposit of fat upon the surface of the heart, and the left ventricle contained some decolorized fibrin. The valves were all in a normal condition. An aneurism, of the size and figure of a large peach, had sprung from the transverse portion of the arch of the aorta, extending from the arteria innominata to the origin of the left subclavian, and pressing upon the latter vessel to the extent of an inch beyond its origin. It had arisen from the superior and posterior wall of the vessel, passed backwards to the vertebral column, by which it was indented, and thence to the right side behind the trachea, which had, however, escaped pressure. The œsophagus had been carried into the right pleural cavity by the advancing aneurism, upon the surface of which it was stretched, and firmly attached; it readily permitted the finger to pass through its canal at the seat of compression, and returned lower down to its normal position in the posterior mediastinum. The left carotid sprang from the sac, but was not diminished in calibre; the left subclavian was nearly obliterated as far as the origin of the vertebral artery. The direction of the arch of the aorta was so altered, that the branches arising from its transverse portion held an antero-posterior relation to one another. The right pneumogastric nerve was stretched upon the outer surface of the sac, whilst the left recurrent, extended upon and inextricably involved in it, had been carried over to the right side of the spinal column. The vertebræ were not eroded.

The paroxysms of dyspnœa and stridor, which were manifestly of a compound character, both laryngeal and bronchial, were explained by the state of tension in which the left recurrent and the right pneumogastric nerves were found; excitement of any kind, by increasing the force of the heart's action, must have caused temporary expansion of the sac, and so increased the

tension of these nerves. The position of the tumor was such that the left vertebral ganglion, and the superior dorsal portion of the left sympathetic chain, could not have escaped being crushed by it. Hence, contraction of the left pupil, with ptosis.

FIG. LXXIV.



Aneurism of the transverse portion of the arch of the aorta. Mrs. L. I.

- A Posterior view of aneurism, with œsophagus attached to, and expanded upon, it.
- B Lower portion of œsophagus, with *plexus gulæ*.
- C Descending thoracic aorta.
- DD Pneumogastric nerves.
- E Left recurrent laryngeal nerve at point of incorporation with aneurism.
- F Left subclavian artery, giving off vertebral, and pressed upon by aneurism.
- G Left carotid artery.
- H Cervical portion of œsophagus.
- I Heart seen from behind, and laden with fat.

The lungs, in an emphysematous condition, are shown on either side.

Absence of the physical signs of aneurism must be attributed to the masking of the tumor by emphysematous lungs. The absence of dysphagia, notwithstanding the displacement and flattening of the cesophagus by the sac, is worthy of special notice. I believe this was due to the want of counter-pressure, and of ulceration of the gullet (see remarks at p. 1102).

The annexed engraving, from a drawing by the late Mr. Connolly, represents a posterior view of the parts after dissection.*

CASE CXLI.—*Strain of Back and Chest, followed by Pain and Syncope; Dysphonia, Cyanosis, and great Edema of Upper Half of Body, with Depression of Temperature; Feeble Respiration in Apex of Right Lung, with Dulness, Impulse, Thrill, and Murmur in the same situation; Death by Coma. Autopsy: Edema of Lungs; Aneurism of Ascending Aorta pressing upon Right Bronchus, Right Branch of Pulmonary Artery, and Descending Cava, and opening into the latter vessel, thus forming an Arterio-venous or Varicose Aneurism.*

Michael W., a mason, of intemperate habits, aged thirty-three years, was received into hospital on October 17th, 1865. His health had been failing for the last year, during which he had undergone many privations; five weeks prior to admittance, whilst lifting a heavy stone from the shoulders of a fellow-workman, he made a false step, and strained his back. He complained, at the moment, of acute pain in the dorsal region, and was compelled to relinquish work for some days. He then resumed his labour, and suffered no further inconvenience till a fortnight later, when he experienced a sudden faintness, not accompanied by pain, whilst walking in the street, and would have fallen had he not supported himself against some fixed object. After a few moments this feeling passed away, and he was able to proceed. About this time I saw him as an out-patient at the hospital; he seemed to be under the influence of drink, was hoarse, and had cough. I did not examine his chest on this occasion. Three weeks later he presented himself again, and, owing to the striking peculiarity of his appearance, was at once admitted. The face, neck, and chest were swollen and

* Vide *Proceedings of the Pathological Society of Dublin*, vol. ii., part iii., new series.

deeply cyanosed, the face and neck being of a dark purple tint, as if stained with dilute ink. The eyes were prominent; the conjunctivæ injected with venous blood, and the pupils contracted. The neck was bloated, so that the sub-maxillary and supra-clavicular fossæ were obliterated. The cervical veins were distended, but devoid of pulsation or thrill. The superficial veins of the chest and arms were distended and tortuous, and the finger-ends were livid. The upper half of the body was cold, and the man complained of constant chill, whilst below the diaphragm the surface was of the normal colour and temperature; the feet were warm. Respiration 24; voice somewhat husky and muffled; cough, with mucous expectoration; pulse 114, small, but regular, and not visible; tongue moist and livid; bowels natural; no dysphagia.

The chest was abnormally resonant in front, but less so on the right side from the clavicle to the level of the nipple. Respiration was puerile on the left side; over the lower portion of the right side it was likewise puerile, but above the nipple in front, and the middle of the supra-venous fossa behind, it was feeble. On both sides mucous râles were audible. Precordial dulness was somewhat extended. The heart's action was regular; the impulse was strong, and unaccompanied by *frémissement*; the apex pulsated at the xyphoid cartilage. At the base of the heart two bellows-murmurs were audible. One of these, systolic in rhythm, harsh and loud, was transmitted upwards through the arch of the aorta, and into the carotid arteries; it was diffused over the entire chest anteriorly, but was louder on the right than on the left side, and had its point of maximum intensity at the third right costo-sternal junction, where the second murmur was not audible. It was likewise heard in the right infra-spinous fossa, and was there associated with systolic impulse. The second murmur was diastolic in rhythm, rather faint, and nearly masked by the former; it replaced the second cardiac sound, and was strictly confined to the lower portion of the sternum. A feeble impulse, at the acme of forced expiration, was perceptible beneath the right clavicle.

On the night of the 20th the man was delirious; and, on the following day, nine leeches were applied at the upper part of the

sternum; the leech-bites bled freely, with great relief to the patient, and it became necessary to arrest the hæmorrhage by means of lunar caustic.

On the 22nd, the cedema had increased, and the patient complained of great tightness in his neck; the impulse beneath the right clavicle was now accompanied by a feeble thrill. An active diuretic mixture, including large doses of acetate of potash, had been hitherto given.

On the 23rd, owing to the great increase of lividity, cedema, and venous tension, I deemed it necessary to take blood from the arm; eight ounces were drawn, with great relief to the patient. The superficial veins of the left forearm were inflamed and marked by red lines on the 24th; leeches and poultices were applied. Cyanosis of the upper half of the body was now extreme, whilst the lower half retained its natural appearance. I wished to have a photograph taken, but the patient's consent could not be obtained.

On the 27th, voice was nearly extinct; he complained of an overpowering sense of drowsiness, and died soon afterwards, rather suddenly, in a state of coma.

On examination of the body, the lungs were found to be very voluminous, and engorged with blood and serum; their specific gravity was not, however, much altered. The pericardium, which was completely enfolded by the lungs, was much thickened, and adherent to the heart throughout. The heart was of the normal size and consistence; the right auricle contained a mass of dark blood-clot; the valves were all healthy.

The superior cava, at its junction with the auricle, was flattened from before backwards, and its opposite walls were attached to one another, leaving a minute passage into the auricle at either side; one of these passages admitted the little finger, but through the other a probe only could be introduced.

The aorta was considerably dilated, and its lining membrane was dotted with minute points of atheroma. A large opening, of an ovoid figure, one inch and three quarters by one inch in diameter, extended across the posterior wall of the aorta, half an inch above the valves. The margins of this opening were smooth but irregular; it led into an aneurism as large as a middle-sized

apple. The walls of the sac were thick and smooth, but rugous; it contained some loose masses of fibrin and dark coagulum. The right branch of the pulmonary artery had been pressed upon, and all but closed, by the aneurism, which had likewise, by its advance forwards and outwards, compressed the descending vena cava, and, as above stated, nearly occluded that vessel, from its entrance into the auricle to a point half an inch below the junction of the vena azygos. The last mentioned vessel was greatly enlarged. The sac of the aneurism had communicated with the vena cava by three openings, one of which was as large as a threepenny piece, the others being much smaller. The edges of these openings were thin and jagged, and on the venous side each was bordered by a deep red line. The superior primary branch of the right bronchus had been likewise compressed by the aneurism. Hence, feeble respiration in the upper lobe of the corresponding lung. Dysphonia was most probably due to cedema of the glottis; but this cannot be positively affirmed, as permission could not be obtained to examine any part of the body except the chest.

The diagnosis of aneurism had been made from the following facts and circumstances: The commencement of illness with strain, followed by pain in the back, and faintness; the evidence of pressure upon the superior cava by a tumor, and the existence, at the point where such pressure must have been exercised, of percussion-dulness, feeble respiration, impulse, and thrill. The presence of impulse, also, in the right scapular region, whilst the apex of the heart pulsated at the xyphoid cartilage, proved at once that two distinct centres of pulsation existed, and that the superior of these two occupied such a position, and had acquired such dimensions, as to afford a rational explanation of the venous and bronchial obstruction above mentioned, and, finally, the existence of a harsh systolic murmur, audible in its greatest intensity at the second centre of impulse.

The absence of well marked impulse, positive dulness, and dysphagia, was explained by the position of the aneurism with relation to the lungs and œsophagus.*

* Vide *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., p. 4.

CASE CXLII.—*Hæmoptysis, Dyspnœa, Huskiness of Voice, Stridulous Breathing and Cough; Tumor, Yielding Double Impulse and Sound, beneath Right Sterno-Clavicular Articulation; Feebleness of Right Radial Pulse; Tracheal Respiration at Upper Portion of Sternum; Bronchial Respiration in Right Scapular Region, and Feeble Respiratory Sound at Apex of Left Lung; Great Increase of Tumor, and Dislocation of Clavicle; Neuralgic Pains, and subsequently Œdema of Right Shoulder and Arm, Right Side of Chest and Neck; Contraction of Right Pupil, with Ptosis, and Elevation of Temperature on Right Side; Partial Suppression of Respiration in Upper Portion of Left Lung; Increased Dyspnœa and Stridor; Death. Autopsy: Acute Miliary Tuberculosis of both Lungs; Heart Hypertrophied and Fatty; Large Fusiform Aneurism of Ascending and Transverse Portions of Arch of Aorta, pressing upon Trachea, Left Bronchus, and Descending Cava, Perforating Sternum and Ribs, and becoming Diffused beneath Pectoral Muscles; Sac containing detached Laminated Fibrin.*

Bernard B., aged fifty-four years, a labourer, of intemperate habits, admitted into hospital in February, 1875. Had bronchitis two years ago, and has spat blood. His voice became husky five weeks before admission, and a fortnight later, he noticed the swelling, to be described presently, on the front of his chest. The following was his state on admission: Voice somewhat husky, and respiration slightly stridulous (from below); there was occasional dry cough, of a harsh, laryngeal, and metallic character. Slight dyspnœa, with darting pains in the right mammary region passing round to the axilla. There was neither engorgement nor pulsation of the cervical veins, and no congestion of the face, or fulness at the root of the neck. Pulse 84, regular, and moderately full, but slightly weaker in the right arm than in the left. Pulsation of carotids equally distinct. Between the second and third costal cartilages of the right side, and close to the edge of the sternum, there was a soft tumor of a conical figure, dull on percussion, and tender to pressure; it was about two and a-half inches in diameter at the base, and projected about an inch and a-half above the level of the surrounding surface; it communicated to the hand a double shock, viz.:

an impulse of an expansile character, coinciding with that of the heart, and preceding slightly the pulse at the wrist; and a diastolic impulse of arrest. On auscultation it yielded two sounds closely resembling the normal sounds of the heart, the first, however, being somewhat less pronounced than the corresponding cardiac sound, and coinciding with the principal impulse, whilst the second was sharp and accentuated. There was no murmur. Dulness extended horizontally three inches to the left of the tumor; namely, to a vertical line touching the clavicle one inch beyond its sternal extremity; on the right side it extended as far as the middle third of the clavicle. The *manubrium sterni* was slightly prominent, and respiration was here tracheal. A few engorged cutaneous veins were visible below the tumor.

The pupils were normal and equal; respiration was slightly accelerated, and became stridulous after the effort of sitting up in bed; respiratory sounds were equal on the opposite sides in front, and, with the exception above mentioned, strictly normal; posteriorly, respiration was slightly bronchial in the right scapular region; no dysphagia.

After he had been a few days in hospital, he complained of fixed aching pain in the right shoulder, which subsequently extended up the right side of the neck, above the ear. This was relieved by laudanum fomentations. He was strictly confined to the horizontal posture; was allowed full diet, and four ounces of wine. Two leeches were applied below the tumor, where he complained of sharp pain for one day, and tincture of the perchloride of iron was given in doses of ℥xv thrice daily. A chamois pad was strapped down over the tumor, and a sleeping draught, consisting of grs. xv of chloral hydrate, with ℥x of Battley's sedative, in an ounce of water, was given every night.

March 22nd. The tumor has enlarged and advanced considerably, tilting forwards the sternal end of the right clavicle and the clavicular portion of sterno-mastoid muscle; it has become firmer. Pulse 100, regular, and now equal. (See subjoined tracings, Figs. LXXV. and LXXVI.) Pain in shoulder and neck less troublesome. Acetate of lead, in gr. v doses, thrice daily, was now substituted for the iron previously given. Iodide of potassium was subsequently given, and at a later period phos-

phorus (one-thirtieth grain, thrice daily) was made trial of for a week. The benefit derived was not appreciable, except from the iodide, which relieved his pain.

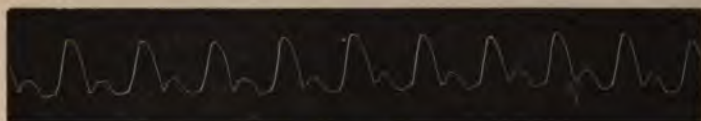


FIG. LXXV.

Aneurism of arch of aorta. Bernard B.
Right radial; pulse 100. March 18th, 1875.

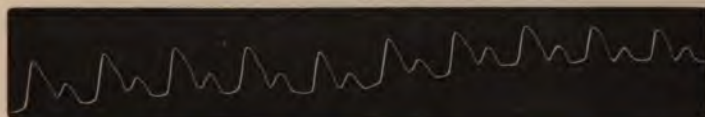


FIG. LXXVI.

Aneurism of arch of aorta. Bernard B.
Left radial; pulse 100. March 18th, 1875.

April 7th. Respiration all but suppressed in the upper portion of the left lung. Tumor now projects more than two inches; it is elongate vertically, appearing behind the sternal end of the clavicle, which is dislocated upwards and forwards, and ascending nearly to the level of the cricoid cartilage. It measures at the base six inches by five, is yielding at all points, and, on the upper portion of the anterior surface, it presents a secondary prominence of a deep purple tint surrounded by enlarged cutaneous veins. In this situation the sac seems to consist of the integument only, and to be on the point of giving way. A double impulse, now very feeble, and a faint double sound unaccompanied by murmur, are still perceptible over the tumor. Pulse not detectable on the right side; in the left arm it is 108, irregular, and extremely feeble. Respiration 24, long-drawn and stridulous, both with inspiration and expiration. Contraction of the right pupil, with ptosis on the same side; oedema of the right arm, and of the right side of face and neck. Temperature in right axilla, 99° F., in left axilla, 98° F. Four ounces of blood were drawn by venesection, whilst the circulation was arrested in the right carotid by means of finger-pressure; the object of the venesection was, to prevent

increased tension of the sac whilst an attempt was being made to effect coagulation of its contents by arresting the circulation through it. This experiment, proving very irksome to the patient, was discontinued after half an hour, a period obviously insufficient for testing its utility. Ice was applied to the tumor with the twofold object of alleviating local tenderness and promoting coagulation in the sac.

8th. Patient was delirious last night; is slightly incoherent to-day; respiration loudly stridulous; ptosis and contraction of pupil, with congestion of conjunctiva, in right eye; temperature in the right external auditory canal, 97° F., in the left, 93.4° Galvano-puncture was now proposed with a view to effecting coagulation in the sac; but the patient, having been candidly told in reply to his inquiry, that the prospect of success from the experiment was not encouraging, declined to submit to it.

9th. Death occurred by slow asphyxia at 9.30 a.m. to-day, respiration having been greatly embarrassed, and attended with loud stridor for some time previously.

The body was examined on the following day. The lungs were studded throughout with disseminated miliary tubercle; at the apex of the right lung there was some thickening of the pleura, with old adhesion, but, with these exceptions, no trace of disease existed in either lung. The heart was somewhat enlarged, and remarkably fatty on the surface; the right ventricle was dilated, its wall was thinned, and in the outer three-fourths of its thickness, composed of fat; the left ventricle was slightly dilated and somewhat thickened; the valves were all healthy. The aortic orifice was normal, and its valves were competent. The ascending portion of the vessel was dilated to the size of a man's wrist; its walls were thick and rigid; and, at the junction of the ascending with the transverse portion of the arch, an aneurism sprang from it, which involved the entire transverse portion as far as the origin of the left subclavian artery. It was of the consecutive false kind, had perforated the manubrium of the sternum, in which an opening as large as a half-crown piece, with thin, irregular, and spiculated edges existed, and had dislocated the clavicle from the sternum. A circumscribed false aneurism, of the dimensions previously stated, had been formed in front of the chest beneath the pectoral muscles and aponeurosis, the

covering of which had been reduced at one point to the integument. The entire of the first portion of the sternum, the inner extremity of the right clavicle to the extent of an inch and a-half, and about an equal length of the first and second costal cartilages, which were completely ossified, projected into the aneurism, and were denuded of periosteum. The extremity of the clavicle, and that of the first rib with a piece of the sternum attached, were quite free within the sac. The lining membrane of the ascending portion of the aorta was of a deep crimson tint, and studded with minute yellow specks of atheroma. The sac was occupied by a firm and laminated shell of fibrin half an inch thick, which formed at its dome a *cul de sac*. This shell had been detached from the sac throughout, manifestly by the entrance of blood at some point of its circumference, and a false aneurism had been subsequently formed by rupture of the sac at its weakest point, under the pressure of the blood so infiltrated. The cavity of the fibrinous shell was smooth, and was occupied by soft loose coagula. The origin of the *arteria innominata* and left carotid were involved in the aneurism. The superior vena cava was stretched upon, and incorporated with, the exterior of the sac on the right side; it was greatly elongated, and its wall was, at two points, reduced to the lining membrane by the pressure of the sac. The left wall of the trachea, immediately above its bifurcation, had been pressed inwards by the aneurism, which formed a prominence within it; the secondary branch of the left bronchus supplying the upper lobe of the lung, had been likewise compressed, and was greatly reduced in calibre. The pneumogastric and recurrent laryngeal nerves of both sides were much thickened. The branches of the superficial cardiac plexus were in a similar condition. The mucous lining of the larynx was highly oedematous, the rima being nearly closed by the surrounding tumefaction. The bronchial glands were enlarged, and of a deep black colour.

In addition to the nine cases given in detail, including Case 69 (p. 759), in each of which a *post mortem* examination of the body was made, I have had under observation, but without the advantage of autopsy, seven other cases of thoracic aneurism. I

propose summarising these cases very briefly; and, for sake of uniformity, I shall do so with special reference to the several heads given in Tables XVI. and XVII.

CASE CXLIII.—William C., aged fifty-four years, a car-driver, of intemperate habits, admitted March 11th, 1868. Seven months ailing. General nutrition good. No history of strain or injury. Aneurism of the transverse portion of the arch, yielding a single and systolic impulse and two sounds, but no murmur. The direction of growth was forward, and there was no valvular disease or enlargement of the heart.

CASE CXLIV.—William R., a porter, aged fifty years, and temperate, admitted March 12th, 1868. Had been accustomed to lifting and carrying heavy weights. General nutrition good. Ailing three years. Aneurism of arch of aorta at junction of ascending and transverse portions, and projecting three inches above the surface at the upper right margin of the sternum. It yielded a double impulse and a double sound, but no murmur. No valvular or other cardiac lesion discoverable. Paroxysmal dyspnoea, with loud stridulous respiration, and harsh ringing cough. Relief of dyspnoea and cough from gr. x doses of the bromide of ammonium, and of pain in tumor from repeated application of ice.

CASE CXLV.—Benjamin W., a porter, had served three years in the army, temperate, and well nourished, accustomed to lift heavy weights. Ten months ailing. Aneurism of descending portion of arch, pressing upon left bronchus. No aneurismal impulse, sound, or murmur. Suppression of breathing in left lung; hæmoptysis; stridulous respiration; metallic cough, and recurrent paroxysmal dyspnoea with loss of consciousness. Death by asphyxia. Examination of body not permitted. Copious discharge of blood from the mouth and nostrils after death.

CASE CXLVI.—Mrs. C., aged thirty-five years, a farmer's wife, admitted May 11th, 1872. Illness commenced suddenly and without assignable cause four months previously, with sharp

pain in left axilla, which was followed by numbness of left arm, of a week's duration. Aneurism engaging left extremity of transverse portion of arch of aorta, projecting on left side of chest, from second to fourth intercostal space, close to sternum, and yielding a double impulse and double sound, with systolic murmur. Sharp shooting pains in left side of chest, left shoulder and arm, as far as elbow. Feeble respiration in left lung, and elevation of temperature, with local perspiration on left side. Double sound, without murmur, in left scapular region. Postponement of radial pulse. Mitral inadequacy. Pain relieved by leeching, and great benefit from \mathbb{M}_{xv} doses of tinct. of the perchloride of iron.

CASE CXLVII.—John F., a farmer of intemperate habits, aged forty-two years, was received into hospital, July 13th, 1872. Five years previously he was thrown from a great height, and his chest came in collision with a stake. An aneurism of ascending aorta, projecting at right sterno-clavicular articulation, and yielding a systolic impulse and double murmur. Inadequacy of the aortic valves (relative ?) and hypertrophy and dilatation of the left ventricle; severe shooting pain in right side of chest and neck, and right arm, relieved by hypodermic injection of morphia; much benefit from \mathbb{M}_x doses of tincture of the perchloride of iron.

CASE CXLVIII.—Mr. William B., an accountant, aged forty-five years, temperate, was brought to me by Dr. Brady, in June, 1873; nutrition satisfactory; no definite history of commencement of illness. Aneurism of ascending aorta, touching anterior wall of chest at right margin of sternum and level of third costal cartilage, within a space equal to a crown-piece in diameter, and yielding a systolic impulse and a double sound, without murmur. Great venous turgescence of face and neck. Paroxysmal dyspnoea with stridor, and increased venous distention produced by the slightest exertion; hæmoptysis. Forward growth of tumor. No valvular disease, or enlargement of heart. I again saw this gentleman with Dr. Hudson, on the 7th of June, 1875. He had had, in the interim, several attacks

of paroxysmal dyspnœa, accompanied by stridor, tumultuous action of the heart, and failing pulse. He had also repeatedly spat blood. The superficial veins of the chest and epigastrium were now enlarged and varicose. The jugular veins were likewise distended, and the face somewhat congested, but cyanosis had disappeared. Respiratory movement was defective on both sides of the chest, and respiratory sound less strong on the right than on the left side; there was persistent dyspnœa, and respiration was accompanied by stridor. The voice was slightly husky. Percussion-sound was now clear on both sides of the chest, and an impulse, with a double sound, was perceptible in the right interscapular space, and at a corresponding point in front. The lower half of the body was quite free from congestion. To inhale M_v of the nitrite of amyl for relief of paroxysmal dyspnœa, and have counter-irritation kept up on the right side of the chest.*

CASE CXLIX.—George D., a labourer, aged forty-four years, of temperate habits, was admitted into the Mater Misericordiae Hospital in December, 1873, and again on the 9th of January,

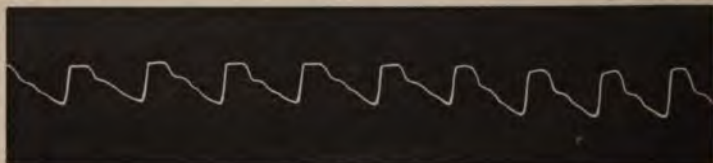


FIG. LXXVII.

Aneurism of arch of aorta. George D.

Pulse 96. January, 1874.

1874, under my colleague, Dr. Hughes, by whose kind permission I was enabled to examine the patient and make notes of the case. Illness commenced without definite cause three years previously. Pains in left side of chest, between scapulæ, and extending up the neck; fulness of left cervical veins; diminished respiration in left lung, and numbness of left arm. Hæmoptysis, aphonia, and dysphagia. Aneurism projecting beneath

* Mr. W. B. died in a paroxysm of dyspnœa while this sheet was going through the press. The body was examined by Dr. Nixon, in presence of Dr. Brady, Mr. Brady, and myself. A large true aneurism, of tubular figure, and quite empty, was found to engage the entire of the ascending portion of the arch of the aorta.

sternal extremity of left clavicle, characterized by a double impulse, and likewise two sounds, the latter of which was sharp and accentuated. The sounds were audible on both sides posteriorly. No evidence of cardiac disease. The annexed tracing of the radial pulse, which was abrupt, but regular, equal in the two arms, and 96 in the minute, was taken by Dr. Nixon. It indicates rigidity of the arteries without hypertrophy of the left ventricle.

CASE CL.—Through the courtesy of my colleague Dr. Nixon, I have been favoured with the opportunity of observing and repeatedly examining a remarkable case of thoracic aneurism, which was admitted into hospital under his care, in January, 1875. The patient, a man aged forty-one years, and intemperate, had been attacked, two years previously, with pain shooting out at the left scapula, and extending round the chest and into the abdomen. He had subsequently repeated paroxysmal seizures of this character. During one of these attacks, and seven days before admittance, the left side of the chest became prominent, and ninety-six hours later, a large tumor appeared in the left scapular region. When first examined in hospital, this tumor occupied the situation of the scapula, which had been displaced backwards and outwards. It was hemispherical in figure, hyperæmic on the surface, and extended from the level of the second to the eighth dorsal vertebra, being fourteen inches in the vertical and nine in the transverse diameter. It yielded a single systolic impulse and a double sound. Pulsation subsequently became visible in the left supra-mammary region, and here respiratory sounds were absent, though elsewhere normal. He had been suffering from bronchitis, with huskiness of voice and cough, which gradually improved as the tumor projected behind, and ultimately disappeared. No dysphagia, or laryngeal phenomena. Precordial dulness was extended. A double murmur existed at apex, with clicking perceptible to the hand, and accompanied by pain and epigastric tenderness. These phenomena, which were due to intercurrent pericarditis, gradually disappeared. On the day after admittance, a slight prominence appeared beneath the sternal end of the right clavicle, and here, percussion-dulness existed over an area three inches in dia-

meter. This prominence yielded a systolic impulse and a rough systolic murmur, the latter of which was transmitted into the transverse portion of the aorta. The second sound was accentuated in the situation of the subclavicular prominence, and, as the stethoscope was moved from this point towards the heart, the murmur gradually faded away.

On the 27th of February, respiration was tranquil, and the cardiac phenomena were normal. Half a grain of morphia was given every third hour to relieve pain, with complete success. This man remained in hospital for several months, during which he was strictly confined to bed. He was supported in the semi-recumbent posture during the day. His diet was generous, but the allowance of liquid was limited. The tumor became solid, and the aneurismal sounds inaudible, but the impulse remained. The man's general health was much improved. He became impatient of further restraint, and left hospital unexpectedly in the end of May.

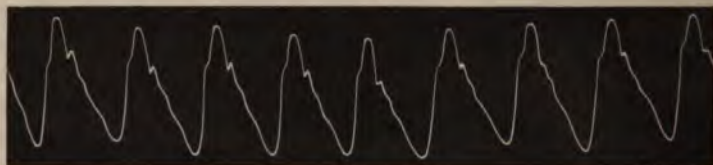


FIG. LXXVIII.

Tracing of posterior aneurism. Peter S. Pulse 84.
February 20th, 1875.

The above tracing from the posterior aneurism was taken by Dr. Nixon, and kindly placed at my disposal.

Aneurism of the Abdominal Aorta occurs most frequently in middle age, and is usually traceable to a definite accident, such as a strain in lifting a heavy weight, or a blow upon the abdomen or back. It is more common in the male than in the female sex. The earliest age at which it occurred amongst the cases given in Table XVII. was eighteen years, and the latest, fifty years; and, of the 16 cases epitomized in that Table, only 1 was in the person of a female.

Of the 59 cases collected by Dr. Crisp, the ages ranged from twenty to sixty years (33 being under forty years), and of this

number only 8 were females.* Since the publication of Crisp's work (1847), seven additional cases in the female have been adduced from British medical records by Dr. A. W. Foot, including one published by himself.† Most probably, however, the ratio, as between the sexes, has not been much if at all deranged within the period mentioned.

The abdominal aorta, in any part of its course, may be the seat of aneurism. The upper portion of the vessel, between the opening in the diaphragm and the origin of the superior mesenteric artery, is, however, its most frequent point of origin. Out of 177 cases of aneurism of the abdominal aorta analyzed by Dr. Sibson, the portion of the vessel at or near the celiac axis was the seat of the disease in 131 instances.‡ The vicinity of the celiac axis may be regarded as the point of election. Hence, the solar plexus is usually involved in, or stretched upon, the tumor, a circumstance to which is due the excruciating pain, radiating through the abdominal viscera, by which aneurism in this situation is distinguished. When situated lower in the abdomen, the aneurism is attended with less urgent symptoms, and is less obscure.

Abdominal aneurism is usually of the false variety, and, as contrasted with thoracic aneurism, is less frequently associated with extensive atheroma of the aorta, and with fatty or other structural disease of the heart. It is likewise unaccompanied by fever, and, with the exception of pain, by less formidable symptoms, and it rarely proves fatal by excentric pressure. The exceptions are, death by exhaustion from excessive pain produced by nerve-pressure or by inanition; from obstruction of the œsophagus, the pylorus, or the thoracic duct.

The symptoms of abdominal aneurism may be now discussed in detail; and first, those which characterize the disease originating in the aorta.

Pain is the most frequent, the most urgent, and usually, the first symptom. It is of two kinds, as pointed out by Dr. Law,§ both of which, however, are not necessarily associated in the same case. The usual pain, and that which constitutes the earliest

* *Structure, Diseases, and Injuries of the Blood Vessels*, p. 161.

† *Dublin Quarterly Journal of Medical Science*, vol. li., February, 1871.

‡ *Medical Anatomy*, pp. 57, 58.

§ *Dublin Journal of Medical Science*, vol. xxi., 1842, and vol. xxv., 1844.

and the most prominent symptom of the disease, is characterized by sudden accessions of the most extreme severity, closely resembling the paroxysms of painter's colic. It radiates from the epigastrium, through the abdomen, into the pelvis, back, and lower portion of the chest; and then, after an indefinite period, usually not exceeding a few hours, quite as suddenly ceases, and leaves the patient exhausted, but free from actual suffering. There is, during the intervals, a total absence of febrile excitement or constitutional disturbance. Accessions of this character are usually determined by causes which produce excitement of the circulation, and increase of vascular pressure. Hence, exercise or emotion, a full meal, copious imbibition of liquids, or the use of stimulating food or drink, is the ordinary cause of an attack. One of the first recorded cases in which pain of this violent and paroxysmal character was associated with aneurism of the abdominal aorta, was observed by Bertin in 1805.* But the most striking example of this kind on record is the well known case published by Dr. Beatty.†

The most urgent pain of this character, is associated with aneurism of the portion of the aorta embraced by the crura of the diaphragm, and has reference rather to the tension produced by the tumor than to the direction of its growth. In this situation, the great splanchnic nerves are exposed to compression, and in a degree proportionate to that in which the aneurism is restrained in its forward and lateral expansion. The constituent branches of the solar plexus, stretched between the semi-lunar ganglia and the coeliac axis, are likewise exposed to tension from an aneurism of the aorta immediately below the diaphragm. Hence, paroxysms of pain radiating through the abdominal viscera, with spasm of the abdominal muscles, and temporary inflation of the bowels, so often witnessed. This pain may be reflected in the course of the ureter, to the testicle or groin, and be readily mistaken for that produced by the passage of a renal calculus. From this, however, it may be distinguished by the absence of hæmaturia.

The second kind of pain above referred to is dull and boring in character, localized, and continuous. It is associated with

* *Opus citat.*, p. 115, observation 42.

† *Dublin Hospital Reports*, vol. vi., 1830.

tenderness at a corresponding point of the spinal column behind, and is all but pathognomonic of erosion of the vertebræ. Not unfrequently connected with this, is a "nipping" pain in the course of the intercostal nerves.* The probability of spinal hyperæsthesia, especially in the female, and the vague and anomalous character of the pain, which must be accepted on the patient's representation, diminish in some degree the value of this symptom. If, however, the pain be aggravated by stooping, or by stamping; by jumping from a height, or by rotating the body whilst standing upon one leg, it may be regarded, if associated with the symptoms and signs of aneurism, as indicating erosion of the vertebræ. In many cases, relief from this pain is obtained only in the prone or stooping posture, and the patient instinctively lies upon his face or leans forward, with his elbows or his head supported in front. Abdominal aneurism located beneath the crura of the diaphragm is rarely unattended by erosion of the vertebral column. With this exception, Laennec was right in asserting that the vertebræ were less frequently eroded by an abdominal than by a thoracic aneurism, owing to the "free surroundings" of the former.†

Jaundice may result from pressure upon the biliary ducts, or venous congestion of the lower half of the body, from compression of the inferior cava. The former of these symptoms is, however, more frequently associated with aneurism of the superior mesenteric artery than of the aorta. Amongst the rare symptoms of abdominal aneurism, whether of the aorta or one of its branches, obstruction of the œsophagus below the diaphragm by the pressure of the tumor;‡ nausea and vomiting from pressure upon the pylorus; forward displacement of the liver, and upward displacement of the heart may be mentioned.

The pulse rarely exhibits any special characters in connexion with abdominal aneurism. It may, however, be intermittent, dirotic, hyperdirotic, or, as pointed out by Dr. W. Wade, even tricrotic; the secondary wave exceeding the first in altitude.§

* *Vide a case by Dr. Law, Dublin Journal, vol. xxii, p. 388.*

† *Opus citat., tom. ii.*

‡ *Vide a case by Sir D. Corrigan, Dublin Journal of Medical Science, vol. ii. January, 1833.*

§ *British Medical Journal, January 4th, 1868.*

I believe that tricotism can exist only in connexion with an aneurism of large capacity, the secondary and major wave being due to reflux from the sac, the walls of which, by their reaction, force a large volume of blood back into the aorta after arterial systole.

The physical signs consist in impulse, dulness, sound, thrill, and murmur. The impulse of abdominal aneurism is single, systolic, and heaving. It may be mistaken for strong pulsation of the aorta from (a) spanæmia, (b) previous hæmorrhage, (c) hysteria, (d) acute dyspepsia, (e) menstrual excitement, (f) the quickening of pregnancy, and (g) intestinal or peritoneal inflammation. It is still more likely to be confounded with an impulse communicated to a tumor overlying the aorta.

For the purpose of arriving at a positive diagnosis, the patient should be placed in the recumbent posture, the anterior abdominal muscles being relaxed to the uttermost by flexing the hip and knee joints. The seat of pulsation should be then carefully explored, by means of the fingers and thumb placed on opposite sides of the aorta, the dimensions of which, and the extent of its pulsation, may be determined by this expedient. If the aorta be found of uniform diameter throughout, and to pulsate equally over its entire length, aneurism will be virtually excluded. It may be necessary to administer chloroform for the purpose of producing complete relaxation of the muscles. In cases of simple pulsation of the aorta, vascular excitement will be found to extend to the iliac and femoral arteries, whereas, the pulsation of aneurism is strictly local; and, finally, under chloroform or other anæsthetic, the former will subside, the latter will not. Sympathetic excitement of the aorta, from inflammatory irritation of the bowels or peritoneum, will be characterized by dysenteric symptoms, or acute sensibility to touch, and by rise of temperature as determined by the thermometer.

The distinction between communicated pulsation, and that of an aneurism, which is often attended with great difficulty, will be more conveniently considered after the physical signs of aneurism have been fully discussed.

An aneurism of the abdominal aorta most frequently springs from the anterior wall of the vessel. When situate, as it usually

is, in the epigastrium, it projects to the left of the middle line in most instances, and, in the progress of growth, has a tendency to *descend*. When discoverable by palpation, it presents in most cases the character of a smooth, elastic, and pulsating tumor, communicating to the hand alternate movements of lifting, combined with uniform expansion and progressive tension, and of subsidence with relaxation. The pulsation is almost invariably single, and slightly posterior in time to that of the heart and the carotid arteries, but synchronous with the radial pulse;* it is occasionally accompanied by thrill. Pressure upon the aorta below the tumor will increase the force of impulse, diminish or abolish the thrill, and arrest the collapse. In a case of thoracico-abdominal aneurism mentioned by Stokes, in which the heart had been displaced upwards and to the right by the pressure of the tumor, the latter yielded a double pulsation and sound, both probably communicated from the heart.† In a few instances the tumor has been hard, rugged, and non-expansile, the sac having been uneven on the surface, and lined by solid laminated fibrin.‡ It may yield no pulsation whatever, even though containing fluid blood, a circumstance which Pirogoff would attribute to the impaction of a solid clot in the mouth of an aneurism with a narrow aperture, or to the compression of the vessel by the aneurism itself, on the proximal side of the sac.§

The evidence elicited by percussion is less conclusive in regard to abdominal than thoracic aneurism. This is due to the deep situation of the tumor, with the intervention of the stomach or intestines in front, and to the presence of the mass of lumbar muscles behind. If, however, the anterior muscles be relaxed, and the stomach and intestines free from flatus, percussion of the tumor will elicit absolute dullness.

Single or double sound, as distinguished from murmur, is rarely heard in front in connexion with abdominal aneurism. It did not exist in any of the cases comprised in Table XVII.

* *Vide* Case 137.

† *Opus citat.*, p. 618.

‡ *Vide* a case by Dr. James Little, *Dublin Journal of Medical Science*, October, 1872; also a case by Dr. James S. Hughes, *Medical Press and Circular*, September 10th, 1873.

§ Quoted by Holmes, *St. George's Hospital Reports*, 1875.

Though not audible in front, sound, usually double, is occasionally heard over a limited portion of the dorsal or lumbar spine. When so limited, and associated with other evidence of aneurism, it is of the utmost diagnostic value.

Thrill is a rare phenomenon in abdominal aneurism. When present, it coincides with the impulse, and is due to vibration of the walls of the sac. Murmur is, next to impulse, the sign most frequently connected with aneurism of the abdominal aorta. It is single, postsystolic, and prolonged, and in quality soft and blowing. It is not propagated in the course of the aorta. If the aneurism arise from the anterior wall of the vessel, the murmur may be audible only in front; and if, from its posterior wall, it may be heard only at a corresponding point in the back. When the aneurism is large, presenting a perceptible tumor in front, and pressing at the same time upon the vertebral column, the murmur will most probably be heard both in front and behind. It occasionally happens that, whilst a murmur is audible in the epigastric or umbilical region, a single or double sound, without murmur, is heard over the corresponding portion of the vertebral column. A few cases are on record in which an aneurism of the abdominal aorta has yielded a double murmur. One such has been published by Dr. Alexander Fleming; in this case a systolic and diastolic murmur were audible at the epigastrium, and a systolic murmur with two sounds in the left loin.* Dr. Willoughby Wade has also published a case in which a double murmur was associated with a single impulse.† The quality of the murmur is sometimes sharp or musical, or it may be shrill in one portion of the tumor, and blowing in another. It is occasionally of an intensely buzzing character, the sac being lax and devoid of coagulum, and therefore capable of strong vibration.‡

The murmur of abdominal aneurism is singularly influenced by posture. As first noticed by Sir Dominic Corrigan, it is suspended when the patient assumes the erect posture; an effect which he attributes to tension of the sac by the weight of the

* *An Inquiry into the Physiological and Medicinal Properties of the Aconitum Napellus*, 1845.

† *British Medical Journal*, January 4th, 1868.

‡ *Vide* a case by Dr. Roberts Archer, *Irish Hospital Gazette*, January 1st, 1875.

column of blood above it. He adds, however, that murmur will not exist, even in the recumbent posture, when the sac is lined by thick layers of fibrin, and therefore incapable of reacting upon its contents.* Cessation in the erect posture is an all but invariable characteristic of this murmur, and the theory propounded by Sir D. Corrigan in explanation of it, is most probably correct. In a few recorded cases, however, murmur has been heard both in the recumbent and the erect posture; and in a case of large abdominal aneurism recently published by Staff-Surgeon Adsetts, a loud murmur was heard in the erect posture, which ceased when the patient lay down.† These anomalies have reference, most probably, to special conditions of the sac and its contents, upon which, in the present state of knowledge, it would serve no useful purpose to speculate.

Abdominal aneurism may be *latent* in regard to physical signs, whilst associated with severe paroxysmal pains, eminently suggestive of aneurism.‡ In such cases a positive diagnosis cannot be made, and an aneurism of small or medium size, arising from the posterior wall of the aorta, will be found to have eroded the vertebral column.

True aneurism of the abdominal aorta, which is of extreme rarity, may be unattended by either signs or special symptoms. A case of true aneurism of the aorta, above the bifurcation, of the size and shape of a pear, in which no tumor could be detected and no pain or tenderness existed, has been published by Dr. Arthur W. Foot. Rupture of the sac was announced by sudden aching pain in the left iliac region, with cramps and pain in the lower extremity, followed by the appearance of a large and rugged tumor in the left iliac, lumbar, and hypochondriac regions. Copious hæmorrhage had taken place behind the peritoneum.§

The *diagnosis* of aneurism of the abdominal aorta may be now briefly considered, with reference to the preceding symptoms and signs viewed in the concrete.

* *Lancet*, April 11th, 1829, and *Dublin Journal of Medical Science*, vol. ii., January 1st, 1833.

† *Irish Hospital Gazette*, April 15th, 1874.

‡ *Vide* a case by Dr. Holmes, *St. George's Hospital Reports*, 1875, p. 191.

§ *Dublin Quarterly Journal of Medical Science*, vol. li.

It is most frequently presented as a smooth pulsating tumor in the epigastrium and to the left of the mesial line, exhibiting a downward growth, and not movable by manual pressure, or by forced inspiration. The symptoms most frequently exhibited are, pain of the twofold character previously described, and local tenderness; whilst the usual physical signs are, a single impulse accompanied by a soft and prolonged bellows-murmur, both slightly posterior in time to the impulse and the first sound of the heart. In exceptional instances a second murmur, and still more rarely, a second impulse of diastolic or postdiastolic rhythm, has been detected. In the back, a single or double sound, without impulse or murmur, are the signs usually exhibited. But a single impulse and a single murmur of postsystolic rhythm, the latter usually associated with a double sound, and not necessarily identical in tone or quality with the corresponding murmur at the epigastrium, are in many cases discoverable over a limited portion of the vertebral column. In a case mentioned by Dr. Stokes, the patient experienced relief from pain when he lay on either side, whilst the tumor and the impulse became imperceptible; when he turned upon his back, he was immediately again attacked with pain, the tumor and the impulse at the same time gradually returning.*

The murmurs, both epigastric and dorsal, are all but invariably suspended in the erect posture, whilst they are usually in no degree influenced by the prone position,† as pointed out by Dr. Stokes, and more recently urged by Dr. William Moore.‡ As regards general symptoms, aneurism within the abdomen is characterized by absence of constitutional irritation. Febrile reaction is of rare occurrence; it is exhibited only towards the close of protracted cases, and is then of a low or irritative character, associated with wasting from constant pain, and loss of appetite and sleep.

The diseases and conditions for which aneurism in the abdo-

* *Proceedings of Pathological Society of Dublin*, December 14th, 1844.

† In a case of abdominal aneurism published by Dr. James S. Hughes, to which further reference will be made, both the impulse and the murmur ceased in the prone position; see *Medical Press and Circular*, September 10th, 1873.

‡ *Dublin Quarterly Journal of Medical Science*, vol. xlix., 1870, p. 479.

men is most likely to be mistaken, are: (a) cancerous or other tumor, receiving a pulsation from the aorta; (b) an engorged liver, receiving a reflux pulsation from the right ventricle through the inferior cava (Burns*); or an enlarged left hepatic lobe, exhibiting the same phenomenon from contact with the aorta (Stokes); (c) an enlarged pancreas similarly circumstanced (Stokes, Burns); (d) psoas abscess; and (e) cystic tumor (Holmes, Pirogoff,† Bakewell‡).

A cancerous tumor in contact with the aorta, and exhibiting a communicated pulsation, has been frequently mistaken for aneurism. Dr. O'Ferrall has reported a case in which a malignant growth in the epigastrium, situate behind the aorta, gave rise to murmur and strong pulsation, similar to those of an aneurism.§

I have met with a somewhat similar case. A man, aged fifty-six years, suffered from severe and continuous pain in the epigastrium, with occasional hæmatemesis; a pulsating tumor, with a loud bellows-murmur, existed in the seat of pain. The patient was emaciated, and died from repeated hæmorrhage. The tumor was found to consist of scirrhus of the pancreas. The duodenum, where in contact with the pancreas, had been ulcerated through, and an opening had been formed in the pancreatico-duodenalis artery, from which the hæmorrhage had proceeded.

Aneurism, on the other hand, is liable to be mistaken for a cancerous tumor. Dr. James Little has published the case of a woman, aged forty-nine, in which a large aneurism of the aorta above the origin of the renal arteries, presented the character of a hard non-expansile tumor, which was lifted *en masse* with each pulsation of the vessel. The difficulty of the case was still further enhanced by the existence of open cancer of the cheek, of which the patient ultimately died. Most of the symptoms and signs of aneurism were, however, well pronounced, and the diagnosis of aneurism was made.||

In the case published by Dr. James S. Hughes, already referred to, there were, in addition to the cessation of impulse and

* *Observations on the Diseases of the Heart*, 1809, p. 270.

† Quoted by Holmes, *loco citat.*

‡ *Royal Medical and Chirurgical Society*, October 27th, 1874.

§ *Pathological Society of Dublin*, January 19th, 1850.

|| *Dublin Journal of Medical Science*, vol. liv., 1872.

murmur in the prone position, the further misleading features of a hard and rugged tumor, and a family history of cancer. Nevertheless, the diagnosis of aneurism was made.

The special characteristics of cancer may be now briefly noticed.

There is, not unfrequently, a family history of cancer, and evidence of the disease on the exterior of the body. The symptoms are steady in progress, and not much, if at all, modified by treatment. There is a tendency to upward, rather than downward growth of the tumor; the pain is seldom severe, and never paroxysmal; the patient is emaciated at an early period of the disease, and is singularly apathetic throughout. Finally, varicose enlargement of the superficial veins, and ascites, are ordinary symptoms, friction-sound may be occasionally detected, and the associated murmur is not influenced by change of posture. With the exceptions previously noticed, aneurism contrasts with cancer in respect of all the foregoing characteristics.

An aneurism which has become diffused by rupture of the sac, may closely resemble a psoas abscess; the extravasated blood, having infiltrated the retro-peritoneal tissue, may present a fluctuating tumor in the lumbar region, or the groin, where psoas abscess usually points. The existence of localized pain and tenderness of the spine will tend still further to mislead. Diffused aneurism, however, usually retains a faint impulse and murmur, whilst femoral pulsation on the affected side is diminished or suspended from pressure of the effused blood upon the artery at some point below the aneurism, or from blocking of the common iliac by a displaced fragment of clot at the moment of rupture of the sac, as occurred in the case published by Dr. Foot, and previously referred to. Interrupted circulation in the lower extremity may likewise serve to distinguish the pain and collapse due to rupture of an aneurism, from the same symptoms when caused by perforation of the stomach or intestine, as shown by Dr. Stokes.

Mr. Holmes has recorded an example of medullary cancer of the kidney, which yielded visible pulsation in the loins, with a loud *bruit*. A correct diagnosis was, however, made from the twofold circumstance, that the pulsation was not in proportion

to the size of the tumor, and that the murmur did not possess the "rushing" character of that of an aneurism.*

The celiac axis and its primary branches, and the *superior mesenteric artery*, are frequently the seat of aneurism. In regard to differential diagnosis, aneurism of these vessels is related to that of the abdominal aorta, as innominate aneurism is to that of the arch. It therefore demands a brief notice here. Examples of superior mesenteric aneurism have been published by Hughes Bennett,† Gairdner,‡ Elliotson, Douglas,§ O'Ferrall,|| and Dr. Arthur Wilson (two cases).¶

In some of these cases, a physical examination had not been made, and the disease was identified only after death; but in others a superficial pulsating tumor, yielding a bellows-murmur, and characterized by great mobility, and by change of position as the patient turned from one side to the other, was recognized. Severe pain in the epigastric and umbilical regions existed in these cases, and in most of them, likewise, jaundice, more or less pronounced, from pressure upon the hepatic or the common biliary duct, was exhibited. In the case published by Dr. Gairdner, together with jaundice, two of the symptoms of gastric ulcer existed; viz., vomiting after meals, and repeated hæmatemesis. Nearly two years later, death occurred by rupture of the sac into the peritoneum. It was then found that long previously the aneurism had opened into the duodenum, whence the hæmatemesis had proceeded.

In a case of aneurism of the hepatic artery recorded by Dr. Stokes, hæmatemesis and persistent jaundice were likewise prominent symptoms; the former, as in cirrhosis of the liver, obviously by exhalation of blood into the stomach, as no direct communication between the sac and the alimentary canal existed; and the latter, from pressure upon the common bile-duct. Death was caused by rupture of the aneurism into the peritoneum. The liver was found to be reduced in volume, whilst

* *Loco citat.*

† *The Principles and Practice of Medicine*, second edition, p. 577.

‡ *Clinical Medicine*, p. 495.

§ Quoted by Gairdner, *opus citat.*

|| *Proceedings of Pathological Society of Dublin*, March 16th, 1850.

¶ *Medico-Chirurgical Transactions*, vol. xxiv., p. 221.

the gall-bladder and the hepatic ducts were distended with bile.* In one of Dr. Wilson's cases, nearly all the structures and fluids of the body were deeply bile-stained, and the biliary ducts throughout the liver were enlarged to the size of the cystic duct; in the other, hæmatemesis and melæna were the most prominent symptoms.

From the evidence furnished by the preceding cases, it would appear that the distinctive characteristics of aneurism of the primary branches of the abdominal aorta, are, mobility of the tumor, and the symptoms of pressure upon (a) the bile-ducts, (b) the portal vein, and (c) the stomach, pylorus, or duodenum.

The duration of life with abdominal aneurism is quite indefinite, and no less variable than in the thoracic form of the disease. In the 16 cases tabulated (Table XVII, p. 1216), it ranged from fifteen days to eleven years.

Death is most frequently caused by rupture of the sac, and effusion of blood behind the peritoneum, into the peritoneal cavity, or into the left pleura. In 7 out of the 16 cases just referred to, a secondary diffuse false aneurism was formed by rupture of the sac behind the peritoneum, the patients having survived this accident for periods varying from a few hours to several weeks. In one of these cases (No. 8) there was a secondary irruption of blood into the cavity of the left pleura.

Extravasation into the left lung or pleura occurred in 3 instances, including that just mentioned, and into the cavity of the peritoneum in 1. Death resulted from syncope without rupture of the sac in 2 cases, from exhaustion in 1, and from Bright's disease in 1.

Of the 47 cases of abdominal aneurism collected by Crisp,† in which the mode of death is stated, 11 opened behind the peritoneum, 10 into the peritoneum, 5 into the left pleura, 3 into the inferior cava, 1 into the lung, 1 into the colon, 1 into the pelvis of the kidney, 1 into the posterior mediastinum, 1 "into the chest." In 13 instances death occurred without rupture of the sac.

* *Dublin Journal of Medical Science*, first series, vol. v.

† *Opus citat.*, p. 168.

The treatment of abdominal aneurism should be conducted on the same principles as that of the thoracic form of the disease. The medicinal agents already mentioned, as useful in the treatment of thoracic aneurism, whether with a view to alleviation or cure, are equally available here.

For relief from pain, large doses of opium or morphia are demanded, and may be given in gradually increased quantity, without fear of narcotism. In one of Dr. Little's cases, already referred to, *twelve and a-half* grains of the acetate of morphia were administered twice daily by hypodermic injection, that is, *twenty-five* grains a-day for some time, without inducing narcotism.

In a case of thoracic aneurism reported by Assistant Surgeon Boileau, which opened into the right bronchus, the patient, an old soldier, took half an ounce of laudanum daily for fifteen days, during which he survived the first hæmorrhage.*

An example of cure of an abdominal aneurism in twenty-five days, under treatment with the acetate of lead (three grain doses, with a quarter of a grain of opium, thrice daily), aided by rest in the recumbent posture, and a restricted diet, has been reported by Dr. Daly.† I incline to think that rest, rather than the medicine used, should be credited with the favourable result of treatment in this case. The success which, in the hands of Mr. Tufnell, has attended the treatment of aneurism by means of rest and regulated diet, without specific medication, would seem to warrant this conclusion. This plan of treatment, which has been already discussed (p. 1141) is no less applicable to abdominal than to thoracic aneurism.

The treatment by compression has, in several instances, been attended with the most gratifying results. The first recorded example of success by this means, was published by Dr. William Murray, of Newcastle-on-Tyne, to whom the great merit of having introduced this mode of treatment, as applied to abdominal aneurism, belongs.‡ In this case, an aneurism of the abdominal aorta involving the origin of the inferior mesenteric

* *Army Medical Report*, 1867, p. 325.

† *Biennial Retrospect of Medicine and Surgery*, 1865-6, p. 305.

‡ *Medico-Chirurgical Transactions*, vol. xlvii., 1864; and *The Rapid Cure of Aneurism by Pressure*, 1871.

artery, was cured by proximal compression of the aorta, the patient being under the influence of chloroform, within the short period of one hour. Two *séances* were given; the first, which lasted two hours, was unsuccessful; but the second, three days later, and continued for five hours, during the last hour of which circulation through the sac was completely arrested, resulted in perfect solidification and cure. The patient died six years afterwards, and both the diagnosis and cure were verified by dissection. The instrument used in this case was the ordinary horse-shoe tourniquet. A second case, in which cure was effected by compression in three quarters of an hour, has been reported by the same physician.* Dr. Murray has likewise mentioned a case treated by Dr. Heath, in which cure by compression was effected in *twenty minutes*.†

Doctor Moxon and Mr. Durham have published a successful case. Compression by means of Lister's "abdominal tourniquet" was maintained under the influence of chloroform for ten hours and a-half; gradual solidification of the aneurism followed, and in little more than a month after the application of pressure, the pulsation of the sac had quite ceased. Four months later the patient was in good health, and the remnant of the aneurism, reduced in size, solid, and free from pulsation, was felt.‡

With a view to effecting immediate coagulation in the sac, as the most favourable condition for solidification of its contents, Dr. O'Ferrall has recommended that distal compression should precede and accompany proximal compression of the artery.§ In a communication of great interest and value, Dr. Mapother has reported the successful application of this principle in his own practice. In a case of large ilio-femoral aneurism in which proximal compression alone had failed, pressure applied simultaneously both above and below the aneurism under the influence of chloroform, effected a perfect cure in four and a-half hours.|| The instruments used by Dr. Mapother were those

* *British Medical Journal*, October 5th, 1867.

† *Ibid.*

‡ *Medico-Chirurgical Transactions*, vol. lv., 1872.

§ *Dublin Medical Press*, March 15th, 1865.

|| *Ibid.*, March 29th, 1865.

known as Skey's and Signorini's tourniquets. In a case of right ilio-femoral aneurism published by Mr. Holden, pressure by means of Lister's tourniquet upon the aorta at its bifurcation, but, as in Dr. Mapother's case, bearing mainly upon the right common iliac, effected solidification of the contents of the sac in four hours.*

For treatment by proximal compression, it is necessary that sufficient space shall exist for the application of effective pressure to the aorta between the diaphragm and the aneurism. Where such is not the case, distal pressure, as practised by Mr. Bryant,† may be tried. The pressure must be such as completely to shut off circulation through the sac, with a view to rapid coagulation of its contents. Before it is commenced, the bowels should be well moved, and freed from flatus, and during its continuance, chloroform or other anæsthetic must be administered. The procedure, though apparently harmless, involves some risk of injury to the bowels. In Mr. Bryant's case, death occurred eleven hours after the experiment, from entero-peritonitis produced by the pressure of the tourniquet.

In none of the cases of abdominal aneurism, four in number, which have come under my notice, has death occurred whilst they were under my observation. In the absence of verification by autopsy, a history of these cases, which was that of aneurism of the abdominal aorta in its typical form, would contribute nothing to the advancement of science, whilst it would add to the bulk of a work already too large.

The case published by Dr. James Stannus Hughes, and previously referred to, claims special notice here, both on account of its intrinsic value to science, and of its melancholy associations. The following abstract of the case, the details of which, as given by Dr. Hughes,‡ are well worthy of perusal, will suffice to exhibit its principal features. An eminent surgeon, resident in Dublin, and aged fifty-four, consulted Dr. Hughes in May, 1867, with reference to severe pains in the loins and sacrum, and ex-

* Mr. Eck, in *St. Bartholomew's Hospital Reports*, vii., and *Biennial Retrospect of Sydenham Society*, 1865-6.

† *Medico-Chirurgical Transactions*, vol. lv.

‡ *Medical Press and Circular*, September 10th, 1873.

tending to the groins and thighs, from which he had been only suffering from the previous evening. Ten years prior to the above date, this gentleman, whose habits had been strictly regular, was supposed to be the subject of a gastric affection, which was accompanied with pulsation of the abdominal aorta. For years he had presented the aspect of a person suffering from malignant disease; was wasted, discoloured, and haggard. His elder brother had died of cancer. For several years also he had had, in the abdomen, a pulsating tumor which he desired to conceal, and in regard to which he refused to admit a thought of aneurism. This tumor was of the size of a man's fist, hard and irregular on the surface, and, in the recumbent posture, pulsated with the up and down movement of a solid tumor overlying a large artery, rather than with the general and uniform expansion of an aneurism. It yielded a systolic bellows-murmur, but in the prone position both impulse and murmur ceased.

Three days subsequently to Dr. Hughes's first visit, and a few minutes after the patient had freely manipulated the tumor with the view of convincing his medical attendants "that it was not aneurism," it gave way, and death from hæmorrhage into the peritoneum followed within three or four minutes.

A large aneurism was found arising from the aorta immediately above its bifurcation. It had grown from the anterior wall of the vessel, and was nearly full of laminated and nodular fibrin, which had given it the rugged feel previously mentioned. Its anterior wall was thin, and had given way in the two places. Two of the lumbar vertebræ were in an early stage of caries. The annexed engraving (Fig. LXXIX.), for the use of which I am indebted to the kindness of Dr. Hughes, exhibits the aneurism as presented after dissection.

Dr. Hughes directs special attention to the following, amongst the many features of interest presented by this case; viz., the long duration of the disease; the absence of pain till three days before death; the unusual character of the pulsation, and the absence of pulsation and murmur in the prone position.

Table XVII. has been constructed from the records of the Pathological Society of Dublin.

FIG. LXXIX.



Aneurism of abdominal aorta. (From Dr. James S. Hughes.)

TABLE XVII.]

ANEURISM OF THE ABDOMINAL AORTA.

No.	Author.	Sex, Age, and Occupation.	Habits and Cause of Aneurism.	Duration in Months, and Seat of Aneurism (when stated).	General Nutrition.	Aneur. Murmur.	Aneur. Sounds.	Aneur. Impulse.	Mode of Death.
1	Dr. Law	Gentleman, 38	Temperate; occasional falls in hunting-field	12: Abdom. aorta, posterior wall, at oeliac axis	Good	Systolic	None	Systolic	Rupture into retro-peritoneal tissue
2	Dr. Stokes	Gentleman	..	18: Abdom. aorta between crura of diaphragm	Good, until shortly before death	Systolic	None	Systolic	Rupture into lung
3	Dr. O'Ferrall	Male, 18	Strain in wrestling	Some mts.: Abdom. aorta betw. crura of diaphragm	..	Systolic	None	Systolic	..
4	Dr. Greene	Female	Undefined injury	139: Abdom. aorta near superior mesenteric artery	..	Systolic	None	None	Rupture into retro-peritoneal tissue
5	Dr. Hutton	Male, 50	..	Some mts.: Abdom. aorta low down	..	Systolic	None	Systolic	Syncope
6	Dr. Law	Shoemaker, 33	..	14: Abdom. aorta between crura of diaphragm	..	Systolic	None	Systolic	Rupture into retro-peritoneal tissue
7	Dr. Law	Male	..	Abdom. aorta at origin of oeliac axis	..	Systolic	None	Systolic	Rupture into retro-peritoneal tissue
8	Dr. Law	Labourer, 35	..	36: Abdom. aorta at oeliac axis	..	Systolic	None	Systolic	Rupt. into left pleura & behind perito.
9	Dr. Stokes	Printer, 40	..	12: Abdom. aorta between crura of diaphragm	..	Systolic	None	Systolic	Rupt. into peritoneal cavity
10	Dr. O'Ferrall	Stonemason, 34	..	18: Abdom. aorta just below diaphragm	..	Systolic	None	Systolic	Rupt. into l. lung & pleura
11	Dr. E. Hamilton	Car-driver, 38	Intemperate	Abdom. aorta in middle portion	..	Systolic	None	Systolic	Rupture into retro-peritoneal tissue
12	Dr. Gordon	Labourer in vitriol works, 50	..	15 days: Abdom. aorta and left c. iliac
13	Dr. M'Dowel	Dischgd. soldier, 29	..	14: Abdom. aorta at bifurcation	..	Systolic	None	Systolic	Rupture into retro-peritoneal tissue
14	Mr. W. Stokes	Cattle drover, 42	Temperate; hardship	48: Abdom. aorta below oeliac axis	..	Systolic	None	Systolic	Asthma
15	Dr. W. Moore	Coal-heaver, 31	Fall from height	36: Abdom. aorta below oeliac axis	..	Systolic	None	None	Syncope
16	Mr. Tufnell	Shipwright, 31	..	Some time: Abdom. aorta below oeliac axis	Good	Systolic	None	Systolic	Bright's disease

ANEURISM OF THE ABDOMINAL AORTA.

[TABLE XVII.]

<i>tion of veth.</i>	<i>Disease of Valves.</i>	<i>State of Ventricles.</i>	<i>State of Aorta.</i>	<i>Observations. General State of Heart.</i>
ard and ily	None	R. Normal L. Dilated and hypertrophied	Atheromat. in seat of disease	Slightly hypertrophied.
i	None
d and ard	None	Not hypertrophied. Caused palpitation by tilting forward heart.
i and to	None
i, down- and to	None	Pain in left testis and thigh. No lumbar or abdominal pain. Jaundice by pressure on hepatic duct. Disease at first sus- pected to be renal.
d
ard	Disease at first taken for sciatica.
ard and ily	Constant aching or boring, and occasional darting, pain.
d	Sudden cessation of impulse in epigastrium. Pain ceased and tumor disappeared when he lay on side, but both returned when he lay on back.
i and to	Murmur ceased in sitting posture. Had most ease while lying on back, although last dorsal and first lumbar vertebrae were found eroded.
ard and ward	Fixed aching pain, varied by paroxysms shooting down thighs, to knees.
of vessels	Atheromatous	Dissecting aneurism engaging entire ab- dominal aorta, and left com. iliac, into which stream had re-entered.
d and ard	None	Normal	Atheromatous	Not hypertrophied. Lived ten days after rupture of sac and formation of a second- ary false aneurism.
ard	Aortic	R. Normal L. Dilat & hypert.	Atheromatous	Hypertrophy and dilatation.
d and to	General hypertrophy. Aneurism had obli- terated right renal artery. Right kidney was reduced to one ounce.
d	None	Normal	Atheromatous in seat of aneur- ism	Not hypertrophied. Aneurism cured by position, rest, and a restricted but nutri- tious diet, in thirty-seven days.

Dissecting aneurism, consists in a breach of continuity of the internal and middle coats of an artery, and a separation of these from the external tunic, to a variable extent over the length and circumference of the vessel, or a splitting of the middle coat, by the force of the blood-current. It is of two kinds: that with a single aperture, through which the blood enters the sac, and returns to the artery; and that which exhibits both an aperture of entrance and an aperture of exit.

The designation "dissecting aneurism" was first used by Laennec; but examples of this form of aneurism had been previously published by Morgagni,* Nicholl,† Burns,‡ and Shekelton.§

Doctor Peacock has given an analysis of 19 cases of dissecting aneurism, including 5 not previously published.|| The ages of the patients, ascertained in 10 cases, ranged from twenty-four to eighty-four years; and of 15 in which the sex was stated, 10 were males, and 5 females. Dr. Crisp, in 1847, published a list of 21 cases, of which 14 were females, and 7 males; the ages of the patients varied from twenty-four to ninety years.¶

The ascending portion of the aorta, in the vicinity of the valves, is the usual seat of primary lesion. Such was its position in 10 out of the 19 cases analyzed by Dr. Peacock. In the majority of cases in which the disease is so situate, the coats of the aorta are tunnelled from its root to the arteria innominata, and death occurs by rupture into the pericardium. 15 of the 21 cases mentioned by Crisp terminated in this way; and of these, 7 died suddenly, 7 lived, one to twelve days after extravasation into the pericardium, one a month, and one "several months."

The case published by Laennec** belonged to the first mentioned category, as did likewise most of those since recorded.

The second form of dissecting aneurism, which, as Dr. Peacock truly observes, represents an "imperfect natural cure" of the

* *De Sedibus et Causis Morborum*, Epist. xxvi., § 15, 17, and 21.

† *Philosophical Transactions*, 1762.

‡ *Observations on Diseases of the Heart*, 1809.

§ *Dublin Hospital Reports*, vol. iii., 1822.

|| *Edinburgh Medical and Surgical Journal*, vol. lx., 1843.

¶ *Opus citat.*, p. 167.

** *Opus citat.*, tom. ii., p. 696.

disease, is very rare. Mr. Shekelton, in 1822, published the two first recorded examples of it.* They were obtained from subjects brought in for dissection, and nothing could be learned in regard to their history. In one, the aneurism occupied the last three inches of the abdominal aorta, and was formed by transverse rupture of the two internal coats on the anterior wall of the vessel. The abnormal channel lay in front of the aorta with which it communicated above by a semilunar opening, whilst below it was connected by two openings with the common iliacs; it was lined throughout by a dense layer of fibrin. In the second example, the walls of the left common iliac artery were channelled; the circulation to the lower extremity having been carried on through the abnormal, whilst that of the pelvis took the ordinary course through the internal iliac passage.

A somewhat similar example, but connected with a history of previous illness, was published by Dr. Henderson. The superior opening was situate near the left subclavian artery, and the inferior in the left common iliac.† Dr. Hyde Salter has recorded an example of dissecting aneurism of the ascending aorta with three openings into the vessel, in a girl aged twenty-four.‡ In this case, it would seem that the blood traversed the abnormal channel twice during each cardiac cycle; forwards with the systole, and backwards with the diastole of the left ventricle. I have met with one example only of dissecting aneurism.§

Aneurism of this form presents no special symptoms or signs by which it may be distinguished from ordinary saccular aneurism. Indeed, owing to the circumstance that it is usually cylindrical in figure, and of small diameter, the symptoms of excentric pressure are generally wanting, and the disease, in regard to symptoms, is not unfrequently latent. When, in the progress of a dissecting aneurism, symptoms of a special character are exhibited, they are strictly accidental, and therefore of no positive value for the purpose of diagnosis. Such were hemiplegia and violent pulsation of the carotid artery on the opposite side, in

* *Dublin Hospital Reports*, vol. iii., p. 231.

† *London and Edinburgh Medical Journal*, July, 1843.

‡ *British Medical Journal*, December 31st, 1870.

§ *Vide Case 129*, p. 1177.

the case published by Dr. Todd.* In that case, a dissecting aneurism of the ascending aorta and the arteria innominata, formed by splitting of the middle coat of the vessels into two layers, gave rise to atrophic softening of the right hemisphere of the cerebrum by pressure upon the common carotid artery; it likewise produced, incidentally, the phenomenon of violent arterial throbbing at the seat of vascular obstruction in the neck. In the case detailed by Dr. Hyde Salter, a diastolic murmur was developed at the aortic orifice by the reflux current through the aneurism descending, as he concluded, upon the ventricular surface of one of the sigmoid valves, and so preventing its closure.

Stenosis of the aorta may be either congenital or acquired. In the great majority of cases it is congenital, and situate at or near the junction of the ductus arteriosus. It was so situate in the 17 cases collected by Crisp,† and in 7 out of the 15 by Luton.‡ In the remaining cases of this last mentioned group it was located as follows: at the origin of aorta, 1; in transverse portion of arch (a double constriction), 1; in the abdominal aorta, 3; general coarctation, 3. In 3 of the cases analysed by Crisp, and in 2 of those by Luton, there was complete obliteration of the aorta. In the acquired form, it may result from local arteritis, with deposition and organization of fibrin at the seat of inflammation; or from the impaction and organization of a migratory clot.

As to form, the constriction may be linear, cylindroidal, or valvular.

According to Rokitansky, congenital narrowing near the ductus arteriosus results from perverted development of the branchial arches in this situation.§ Dumontpallier attributed it to extension, into the aorta, of the obliterative process by which the ductus arteriosus is closed;|| whilst Cheevers supposed it might be due to constriction of the aorta from tension of the left recurrent nerve, produced by the gradual ascent of the larynx into the neck with the first acts of inspiration.¶

* *Medico-Chirurgical Transactions*, vol. xxvii., p. 301.

† *Opus citat.*, 1847, p. 31.

‡ *Nouveau Dictionnaire de Médecine et de Chirurgie*, vol. ii., 1865, p. 744.

§ *Pathological Anatomy*.

|| *Gazette Méd. de Paris*, 1857.

¶ *London Medical Gazette*, 1845, p. 187. See also Craigie, *Edinburgh Medical and Surgical Journal*, vol. lvi.

Amongst the consequences of chronic stenosis of the aorta, are: dilatation of this vessel above, and narrowing below the seat of constriction; hypertrophy and dilatation of the left ventricle; and enlargement of the collateral arteries. If dilatation extend to the orifice of the aorta, the valves may become relatively inadequate.

Death may result from rupture of the aorta, cerebral congestion or apoplexy, local inflammation with thrombosis of the aorta, embolism, or from various intercurrent diseases.

The symptoms are inconclusive, owing to the early establishment of a collateral circulation. There is, however, a marked contrast, as to size and force of pulsation, between the mammary, intercostal, and epigastric arteries, engaged in this subsidiary function, and those of the lower limbs. The only acoustic sign noted has been recorded by Blakiston; viz., a postsystolic murmur, loudest at the right second costal cartilage, and transmitted through the aorta.* But a murmur of this site and rhythm might equally depend upon local roughening of the vessel.

Aneurism of the coronary arteries is of rare occurrence. It is, when present, usually associated with atheroma or calcification of the coats of these vessels, and is, therefore, a disease of middle or advanced age. A remarkable example of it in a boy of seven years, has, however, been published by Dr. Gee. In this case, there were three aneurisms; viz., one of the size of a pea at the apex of the heart, another of the same size at the base of the right ventricle close to the origin of one of the coronary arteries, and a third, as large as a horse-bean, in the posterior inter-ventricular sulcus near the base of the heart.†

The disease usually proves fatal by hæmorrhage into the pericardium.

Aneurism of the valves of the heart is less rare than that of the coronary arteries. The aortic valves are usually its seat, but it has been met with also in the mitral valves.‡ A case has been

* Quoted by Walshe (*opus citat.*), whose work may be consulted with great advantage in reference to the literature of this subject. See also the Memoir of Mr. Nixon, *Dublin Journal of Medical and Chemical Science*, vol. vii.

† *St. Bartholomew's Hospital Reports*, vol. vii.

‡ Vide three examples by Mr. Prescott Hewett, *London Medical Gazette*, vol. xi. p. 980.

published by Sir Henry Marsh, in which a large pouch, distended with blood, and resting against the anterior segment of the mitral valve, had been formed in one of the aortic valve-segments; it had ruptured into the left ventricle by three rents. The acoustic signs were those of obstruction and inadequacy of aortic valves.* Crisp refers to two examples preserved in the museum of St. Thomas' Hospital, and in that of Chatham. A case has been also published by Professor Harrison.†

As to the pathology of this affection, Niemeyer thinks it is due to perforation of one of the coats of the valves, with pouching and distention of the other.

Acute aortitis is occasionally met with in connexion with atheromatous change of the coats of the vessel. The ascending portion is its usual seat. It is characterized by extreme vascularity and softening of the *intima*, which may lead to rupture of the vessel. During life, it is indicated by palpitation, dyspnoea, and substernal pain, with a feeling of constriction of the chest. A harsh systolic murmur, transmitted to a distant portion of the aorta from roughening of the *intima* at the seat of inflammation, may be also developed, as in the case recorded by Dr. Parkes.‡

Rupture of the aorta may result from shock or succussion of the chest. When such an accident occurs, the vessel must have been previously unsound. An example, which was probably of this kind, has been published by Dr. Walshe.§ Death by extravasation may be the immediate result of complete rupture of all the coats; or, the external coat remaining unbroken, death may occur at a later period by its rupture, or a dissecting aneurism may be established.

Perforation of the aorta by a penetrating wound, or by a sharp body swallowed and impacted in the cesophagus, is usually fatal within a few minutes.

* *Proceedings of the Pathological Society of Dublin*, November 28th, 1840.

† *Dublin Journal of Medical Science*, vol. xv., p. 298.

‡ *Medical Times*, February 23rd, 1850.

§ *Opus citat.*, fourth edition, p. 575.

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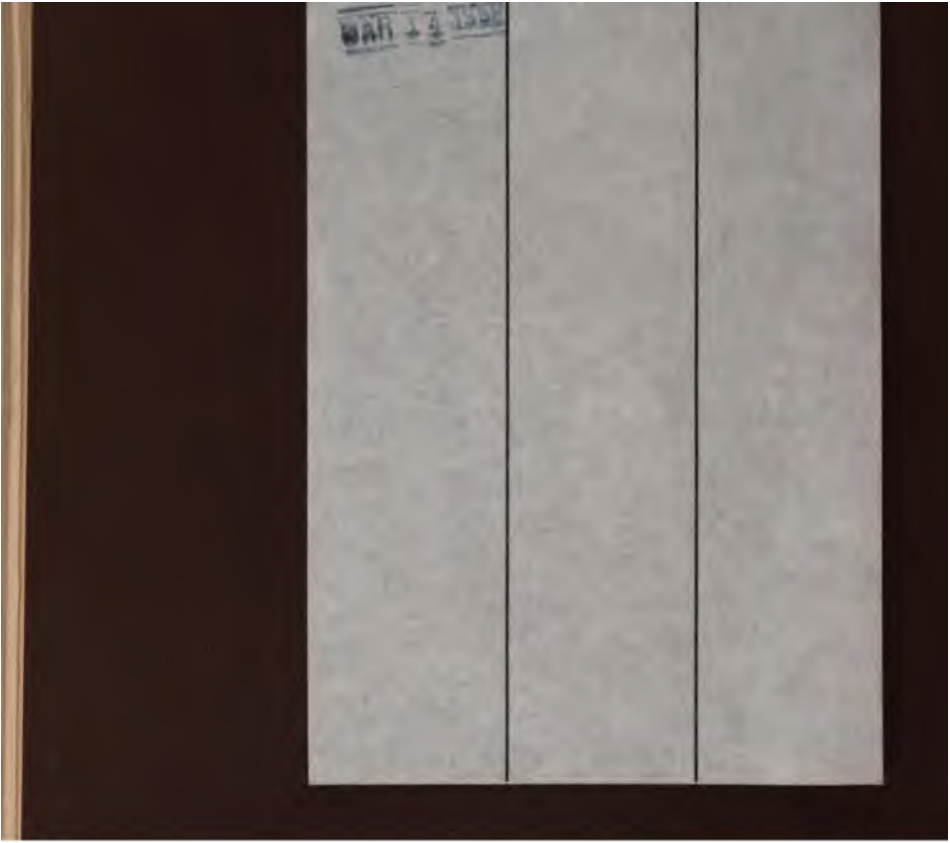
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